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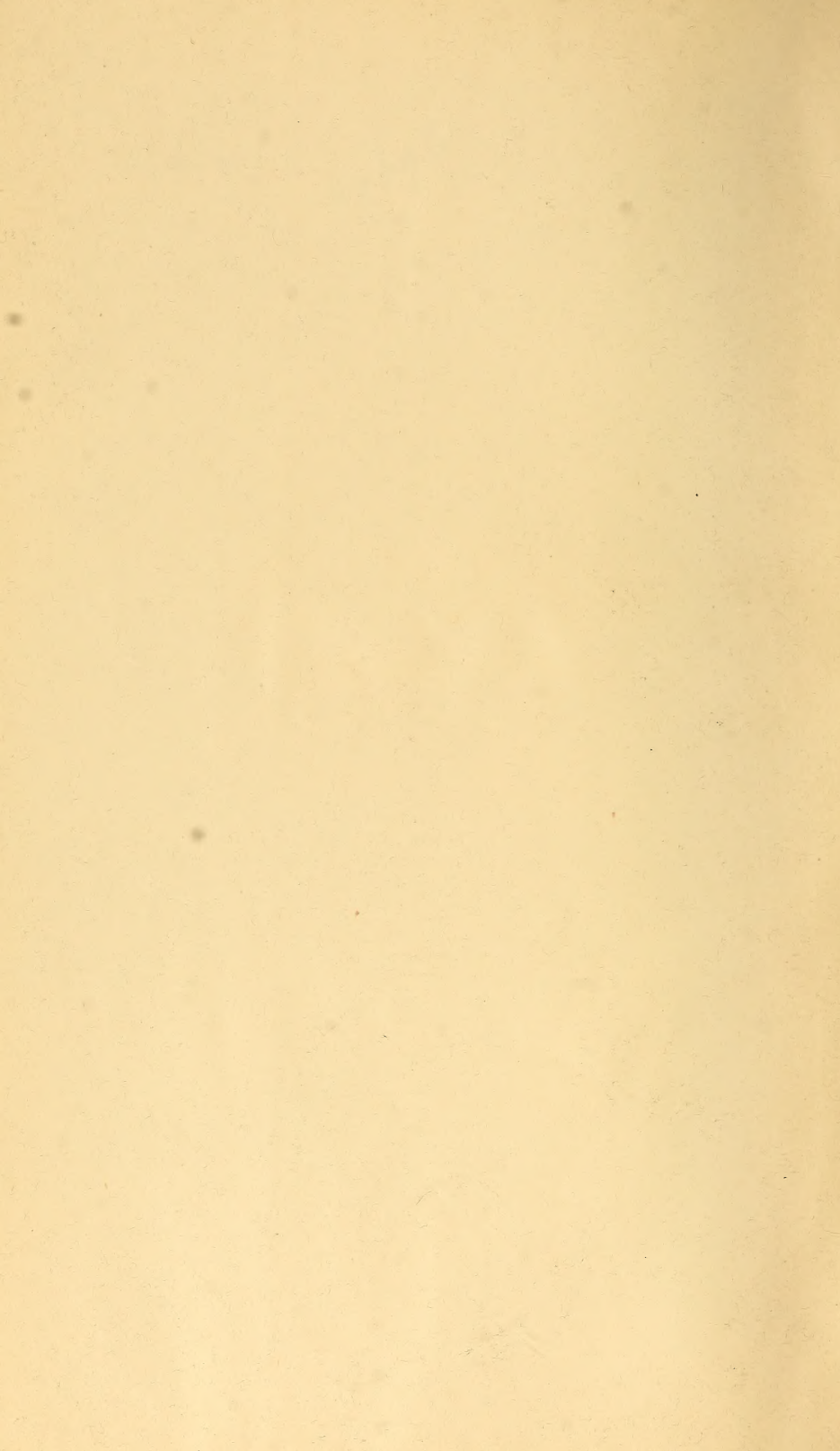




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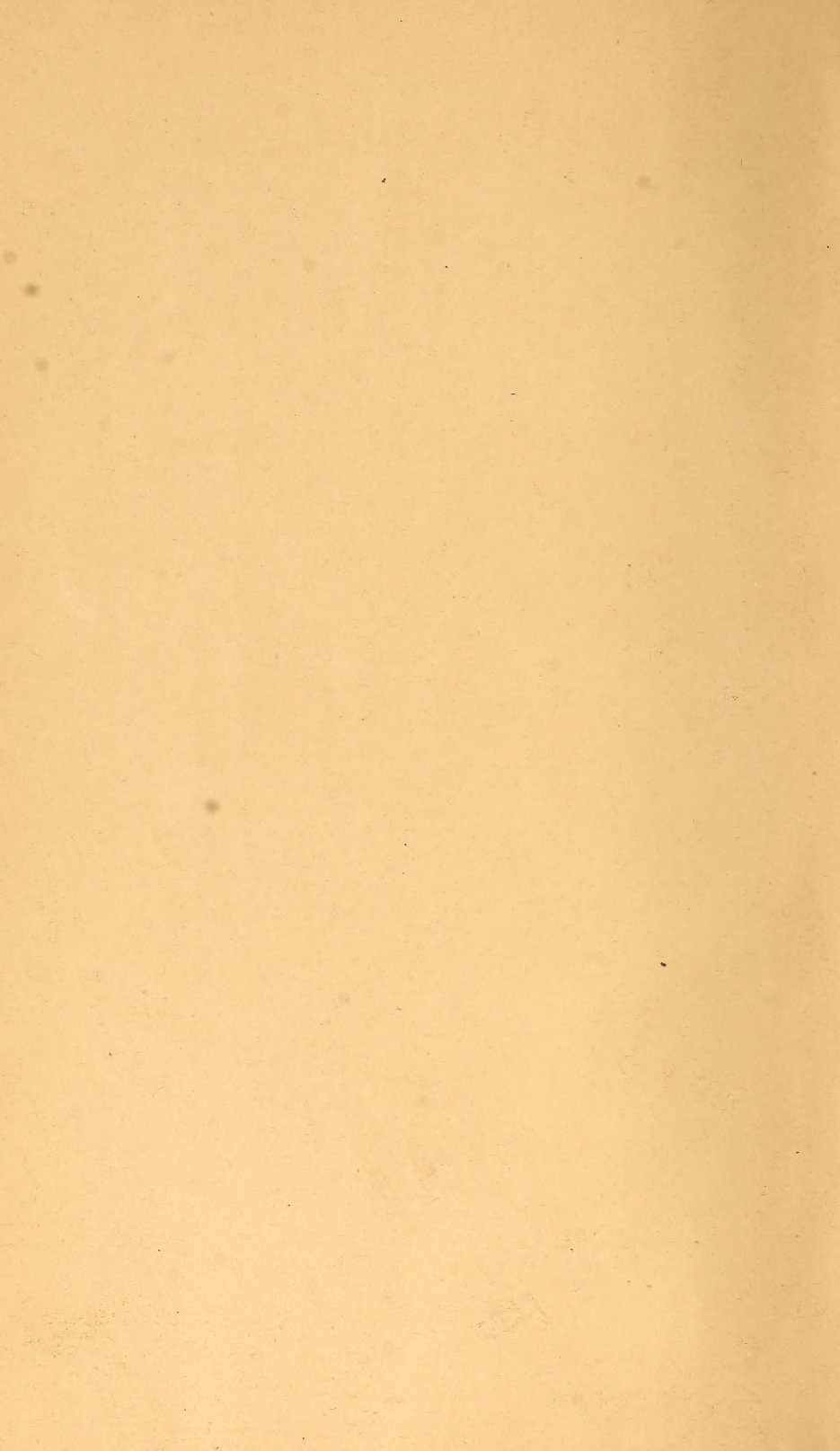














# NERVOUS DISEASES:

THEIR

## DESCRIPTION AND TREATMENT.

A Manual for Students and Practitioners of Medicine.

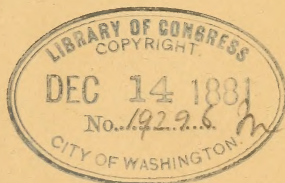
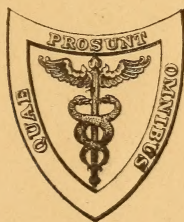
BY

ALLAN McLANE HAMILTON, M.D.,

FELLOW OF THE NEW YORK ACADEMY OF MEDICINE; ONE OF THE ATTENDING PHYSICIANS AT THE  
HOSPITAL FOR EPILEPTICS AND PARALYTICS, BLACKWELL'S ISLAND, NEW YORK CITY; ONE OF  
THE CONSULTING PHYSICIANS AT THE HUDSON RIVER STATE HOSPITAL FOR THE INSANE,  
AND MALE AND FEMALE INSANE ASYLUMS OF NEW YORK CITY, ETC., ETC., ETC.

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TO MY FRIENDS

FORDYCE BARKER, M.D.,

AND

JOHN T. METCALFE, M.D.

AND TO

MEREDITH CLYMER, M.D.,

THE PIONEER

IN THE FIELD OF MODERN NEUROLOGICAL LITERATURE IN AMERICA.



## PREFACE TO THE SECOND EDITION.

In presenting a new edition of my book I wish to express to the profession my hearty appreciation of the favorable reception accorded to the first, which has been out of print for several months. I thank my impartial reviewers, and take pleasure in saying that wherever possible, I have endeavored to adopt their suggestions, and I trust, have succeeded in remedying the faults, many of which are unavoidable in a first edition.

The present edition is enlarged by nearly one hundred pages and contains many new illustrations, in fact this feature of the book has undergone an almost entire change. The enlargement is a matter of necessity, owing to the recent advances in our knowledge of neurological medicine. I have used certain portions of my essay which received the prize of the American Medical Association, in 1879, in the preparation of a chapter upon diseases of the lateral columns of the spinal cord. Other chapters have been remodeled, and I hope improved, especially in regard to the introduction of matter relative to localization of disease in the brain and spinal cord.

ALLAN McLANE HAMILTON,

NEW YORK, 43 EAST 33D ST.

Nov. 1st, 1881.





## PREFACE TO THE FIRST EDITION.

---

It has been my object to produce a concise, practical book; and should the satisfaction be ever accorded me of knowing that I have made the subjects of Diagnosis and Treatment of Nervous Diseases more simple to my readers than I think they now are, I shall be amply rewarded for the task I have undertaken.

I have not considered Insanity, because I believe that this subject deserves much more extended notice than it could possibly receive in a book of this size and kind.

I have deemed it advisable to include a short article upon Cerebro-Spinal Meningitis, though, by many authorities, it is not regarded, strictly speaking, as a nervous disease. I think, if for no other reason, its interesting diagnostic relations entitle it to consideration.

In conclusion, I wish to thank Drs. Loring, Janeway, Mason, Shakespeare, my resident physicians, Drs. Meyer, Naylor, Ryan, and Baldwin, and Mr. F. O. C. Darley, for valuable assistance in the preparation of this volume.

ALLAN McLANE HAMILTON.

NEW YORK, MAY 1st, 1878.





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# NERVOUS DISEASES.

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## INTRODUCTION.

### HINTS IN REGARD TO METHODS OF EXAMINATION AND STUDY.

IN beginning our consideration of the diseases which are to form the subject of the succeeding pages, it is well to start with systematic rules for investigation, and it is of paramount importance that we should pursue some plan which will enable us to avoid confusion, and assist us in making an accurate diagnosis by exclusion. I, therefore, propose a scheme to be used in the examination of patients, and would add a word of caution in regard to the error many of us make in too readily accepting and isolating nervous symptoms as distinct, which, after all, may be expressions of some general disorder. It too often happens that simple digestive disturbances, cholesteræmia, or perhaps uræmic poisoning give rise to symptoms that are seized upon as the basis of a distinct nervous disease, and the error is not recognized in time to arrest the true mischief.

We are to determine the existence and relation of disorders of motility and sensation, as well as mental symptoms, defects of speech, sight, or hearing, together with the causes which enter into their production.

### EXAMINATION OF THE PATIENT.

**PRELIMINARY EXAMINATION.**—Sex, age, temperament, appearance, duration of present disease, existence of complicating maladies, previous history, hereditary predisposition, habits.

### SYMPTOMATOLOGY.

**MOTILITY**, degree of, location of loss or increase (one side or one-half of body?), groups of muscles or single muscles, face, trunk, or extremities, lateral or bilateral, symmetrical or unsymmetrical, loss or exaggeration of electro-muscular contractility, fibrillary contractions, muscular power, associated with deformities or contractures; atrophy or hypertrophy, general or partial; spasms, tonic or clonic, attended or unattended by loss of consciousness; condition of reflex excitability.

*Tremor.*—Local or general, increased or controlled by will, “fine” or “coarse;” time of day, continuous or at intervals; subsidence or continuance during sleep; whether evoked by jarring limb, or by tapping tendons or muscles; increased or stopped by flexion or extension of foot; accompanied or not by pain; associated or not with rigidity of joints when limb is flexed

*Incoördination* of upper or lower extremities, variety of action in which it occurs; gait; aggravation by closure of eyes; loss of muscular sense; loss of locating power.

**SENSATION.**—General or partial anæsthesia; dysæsthesia or hyperæsthesia; susceptibility to painful impressions; temperature; tactile sensibility; sensibility to pressure; pain, localized or general; character of pain, neuralgic, terebrating, dull, or paroxysmal; time when aggravated; its associations; time of transmission of sensation; appreciation of form.

#### DISORDERS OF ORGANS OF SPECIAL SENSE.

*Eyes.*—Nystagmus, strabismus, conjugate deviation (see article Cerebral Hemorrhage), retinal changes, corneal changes, pupillary changes, ptosis, diplopia, amblyopia, amaurosis. The existence of color blindness.

*Ear.*—Deafness, subjective noises, discharge.

**SPEECH.**—Aphasia, slow speech, clumsy speech, ataxic speech, loss of speech (mutism). Visual and auditory relations.

**VERTIGO.**—Variety; concomitant phenomena.

**PSYCHICAL DISORDERS.**—Illusion, hallucination, delirium, mania, melancholia, delusions, and their character, loss of memory, loss of consciousness, imbecility, idiocy, excitability, dementia.

**MISCELLANEOUS.**—Character of cutaneous surface, changes in temperature of general surface or localized spots, cranial temperature, variation in salivary secretions, changes in pigmentation and appearance of hair, perspiration, etc.

#### EXCITING CAUSES; DIAGNOSIS; TREATMENT.

This list, though imperfect, will, I think, enable the observer to pursue a systematic course in examining his patient. He should, at the same time, take careful notes for future reference, so that variations in the symptoms and changes of treatment may be remembered.

Before leaving the subject of examination, I wish to refer to the value of post-mortem examination and microscopical investigation of the morbid anatomical changes. These subjects belong more properly to special works upon pathology and microscopy, but it may not be amiss to add a few hints to those already given in regard to certain important steps to be taken. In removing the calvarium the thickness of the cranial bones should be noted, as well as the condition of the diploë; but extreme care should be employed, in sawing through the bone, not to wound the meninges and brain-substance beneath; for the saw-teeth may unexpectedly tear through, lacerating and injuring these parts, so that they may be almost useless for subsequent examination. After the skullcap has been removed, the

observer should be on the lookout for Pacchonian bodies, and ready to recognize any adventitia that may be attached to the dura mater. The condition of the longitudinal sinus and veins which are contained in the dura mater should be examined as to their fulness, etc.; the thickness, vascularity, color, and opacity of their tissue should also be carefully noted and then an incision may be made, and this membrane slit up with a pair of blunt-pointed scissors, or it may be cut around at the level of the saw cut. The arachnoid and pia mater are then to be inspected: the existence of effusion, either serous, purulent, or bloody; and the presence of granular deposit or vascular changes noted. The brain should be lifted back, and the cranial nerves carefully cut as near as possible to their points of exit from the skull, the optic first, and then the carotid arteries and posterior nerves; next the tentorium, and finally the other nerves, vertebral arteries, and the spinal cord as low down as possible, taking care not to make pressure by insinuating the finger into the foramen magnum. The brain may then be removed.<sup>1</sup> If it is desired to remove the cord, the skin and muscular tissue of the back should be divided and thrown back, and the spinous processes and laminae exposed. These latter should be sawn through on each side and carefully raised by the blade of the chisel. When the brain is removed, it should be placed with the base downwards, and the appearance of the convolutions noted, the membranes having been removed. Evidences of pressure are to be looked for, and the color is to be noticed, as well as the depth of the sulci and superficial evidences of softening or sclerosis, morbid growths, and infiltration. The organ may be turned over, and the arteries at the base inspected in regard to the existence of anomalies, aneurisms, degeneration, thrombosis, or embolism. The fissure of Sylvius may be next examined, and the middle cerebral artery traced by sections. As to the method of making cuttings of the brain, we may, perhaps, find resort to the horizontal section of Flechsig, especially when the patient has presented before death symptoms indicative of degeneration of the internal capsule. We are enabled to carefully compare by this means the relations of the gray nuclei and the peduncular fibres. The cranial nerve-trunks are to be carefully noticed, and if any suspicious appearance is observed, a section may be removed for microscopical examination. The crura and pons are to be examined carefully for softening, secondary degeneration, extravasations and the like, and the appearance of the basal parts of the hemispheres next noticed. The brain-substance may be inspected, in other ways by cutting through the corpus callosum, and turning each hemisphere gently back, or by slicing off the brain-substance with a broad sharp knife previously dipped in water or alcohol, so that the white matter may be examined at different levels, as recommended above. The condition of the ventricles should be noticed as to the effusion of serum or blood, or the condition of the lining mem-

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<sup>1</sup> Removal *en masse*, of the brain and its membranous coverings should never be attempted; the result of such a procedure being mechanical injury, which reduces the organ to a pultaceous mass, rendering it unfit for examination.



branes. The parts at the floor of the lateral ventricles deserve special study, and the corpora striata should be inspected very attentively, the extra-ventricular and intra-ventricular parts being carefully sliced. A vertical section just posterior to the fissure of Rolando (Pitre's section) may be made. The fulness of the vessels in the deep parts of the brain, the existence of patches of softening or induration, and the pressure of cysts, tumors, or morbid growths should be looked for. It is always advisable in cases where aphasia has been a symptom during life, to carefully inspect the anterior convolutions, particularly the third frontal, which is the generally acknowledged seat of the lesion, and we may do this examining at the same time the appearance in the fissure of Sylvius, and carefully slicing that portion of the brain anteriorly, and laterally to the corpus striatum of the left side.

It is hardly necessary to allude to the importance of carefully examining the medulla and the roots of the various cranial nerves, the pyramidal decussation, and the cerebellum, and for this purpose it is advisable to remove such parts as are wanted for subsequent microscopical examination. The cord must be examined critically in cases of spinal disease, and the same directions are given for its inspection. Suspected portions may be cut out and laid aside, care being taken to secure as much of the external roots as possible. In special cases nerve trunks or peripheral nerves may be excised for future examination, and in cases presenting muscular atrophy and degeneration it is well to ascertain the morbid changes in the muscles. If we desire to use the microscope it is generally necessary to harden the tissues, although fresh nervous substance may be teased apart in glycerine or serum by needles prepared for the purpose. If we prefer the first method we may put such masses of the brain or cord as we desire to harden into Müller's fluid, which is prepared as follows:—

R. Potass. bichromat. 50 grammes,  
Sodic sulphate, 20 grammes,  
Water, 1600 grammes :

Or, what is better, the solution recommended by Prof. J. W. S. Arnold, of the Medical Department of the University of the City of New York :

R. Ammon. bichromat. 11 grammes,  
Methyl alcohol, 320 grammes,  
Water, 640 grammes.

Care should be taken not to secure specimens which are too large, as they do not harden thoroughly, the exterior becoming hard while the interior is diffuent and useless. They should be left in the solution for a month or six weeks, but not till they become granular or cheesy, for then it is impossible to make a good section, as the tissue is apt to crumble under the knife. At the end of this time, or when the tissue is quite firm, it may be removed and placed in a fifty per cent. mixture of alcohol and water. The specimen may be examined to test its hardness by making sections with a razor from time to time. If a very thin section can be

made with a moistened razor without parting, adhesion, or crumbling, it may be considered to be in fit condition for removal from the hardening solution. A solution of bichromate of ammonium, 15 grains to the ounce of water, is an excellent hardening solution, in which the specimen may remain until it has been uniformly saturated, and hardening has commenced, and then it is to be removed and placed in a solution of chromic acid, two grains to the ounce of water, where it is to remain until hard enough for cutting. This is the process recommended by Dieters. The specimens may be taken out and kept for use in dilute alcohol till they are needed.

When the hardened tissue is to be examined, it is to be imbedded in pith or paraffine, and either placed in a section cutter, or held in the hand. By practice, this latter procedure becomes quite easy, and very thin sections may be skillfully made. A piece of brain or a length of cord of a convenient size is surrounded by elder pith previously prepared to receive it, and bound in place by a string, or by a piece of fine copper wire. When moistened, the pith swells so that the tissue receives uniform pressure and support. If the paraffine process be that employed, the tissue is to be carefully dried and placed in a small paper mould which is afterwards filled with melted paraffine, this however should not be too hot,<sup>1</sup> and care should be taken to exclude air-bubbles. When cool and solid the upper part of the paper may be torn away, and the specimen is ready for cutting. A flat razor is the best instrument of which I know for ordinary work. Its blade should be dipped in a saucer containing alcohol placed conveniently by, and the face of the section should be moistened from time to time. The individual holding the mould firmly between the thumb, forefinger, and second finger of the left hand, cuts away a portion of mould and tissue so that a level surface is left exposed; then, with moistened razor, he plants the blade, and slowly cuts a thin slice of paraffine and tissue together; this is removed by a camel's hair brush which has been dipped in alcohol, and next dropped into a small vessel containing dilute alcohol, and then placed in the staining fluid, which may be the following:—

R. Carmine (pure), gr. xx,  
 Liq. ammoniæ, q. s. ut dissolv.,  
 Glycerinæ,  
 Aquæ, āā ℥ij.—M.

After being allowed to soak for several hours or days, the sections are removed and dropped into water slightly acidulated with acetic acid. They are now to be placed in absolute alcohol for a short time, and afterwards in oil of cloves until they become transparent. A perfectly clean slide is procured, upon which one of them is placed and a drop (not too large) of Canada balsam is next applied. It is then covered by a thin

<sup>1</sup> I have recently used metallic bottle caps, which may be easily procured. When the paraffine is cool the metal may be stripped off.

glass cover, care being taken to exclude air-bubbles. Various preparations are used to stain nervous tissue; for instance, a solution of chloride of gold will stain the nerve fibres, and render them more distinct; hæmatoxylin and osmic acid are also used, and the black analin process of Herbert Major<sup>1</sup> produces the most beautiful results. These manipulations, however, are out of place here, and I would refer the reader to any one of the excellent text-books that have appeared during the past few years for more explicit directions.

It is often necessary to make sections in all possible directions and positions, and to do this properly the microscopist must not only have practice but patience and care. It is advisable to procure at least two objectives, one for coarse appearances, and the other for minute changes, and I would suggest that these should be an "inch" and a "quarter inch."

#### INSTRUMENTS USED FOR THE DIAGNOSIS OF NERVOUS DISEASE.

It is essential that we should possess certain instruments which shall be more valuable and exact than our unaided senses, so that we may not only make reliable investigations, but compare from time to time such variations as may occur in the patient's condition.

Those I propose to describe are intended for examinations of temperature and sensory changes, and for the detection of altered motility.

**THE THERMOMETER.**—There are several instruments made for the purpose of determining variations in temperature, and though some are of extreme delicacy, I do not think it will be worth while to recommend them, as they are bulky and troublesome, and are better adapted for experimental purposes than actual clinical use, and among these is Lombard's instrument.

In Dr. Seguin's surface thermometer we possess an admirable little instrument for testing the surface temperature. It has an expanded base, and may be applied to the surface of the body, taking care to cover the top by a perforated piece of thin rubber or leather. A coat or two of shellac varnish to the upper part of the bulb will answer the same purpose, viz., that of preventing the mercury from being affected by the temperature of the room. For the determination of deep temperature we may avail ourselves of any of the good self-registering instruments. Two surface thermometers should be used, one on the sound, and the other on the affected side of the body, and the deep temperature may be taken at the same time for comparison. A new form of surface thermometer has recently been made in England. The glass tube is spirally coiled upon itself and enclosed in a circular box. This form has the merit of being unaffected by other than the body temperature.



Dr. Seguin's  
Surface  
Thermo-  
meter.

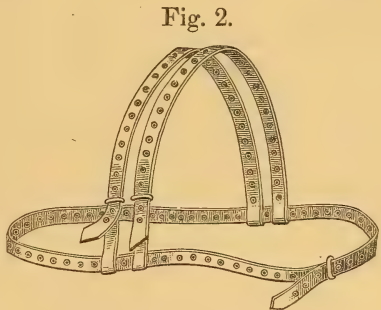
<sup>1</sup> West Riding Reports, vol. v.



Within the past two or three years a great deal of interest has been excited by the remarkable investigations of Broca, who found that it was possible to detect deep changes of temperature in the cerebral organs by means of surface thermometers applied to the exterior of the cranium<sup>1</sup>. Broca's observations were confirmed by those of <sup>2</sup> Dr. Landon Carter Gray, of Brooklyn, N. Y., and by <sup>3</sup> Maragliano and Seppilli, two Italian experimenters. Albers of Bonn was undoubtedly the first person (1861) to suggest cerebral thermometry; but Broca's work was the first undertaken in a systematic and fruitful manner.

By the use of six or more thermometers applied to the head at various points, with every allowance for external disturbing agencies and sources of error, it is found that the central temperature undergoes various modifications, amounting sometimes even to several degrees; and Gray was enabled to diagnose and localize the existence of a cerebral tumor by this diagnostic means. The thermometers should be those known as Seguin's, or, better still, of the form modified by Dr. Gray. They should be tempered perfectly, and so constructed that ordinary pressure upon the bulb shall cause no rise in the column of mercury.

A proper system of straps (Fig. 2), such as has been devised by Dr. Gray, or a cap of gum-rubber, with perforations, enables us to apply the thermometers upon both sides of the head, over the points we desire to examine. Dr. Gray has adopted the names *Frontal*, *Parietal*, and *Occipital*—stations relating to the positions indicated by the names to designate the places over which the tests are to be made. A thermometer is to applied (after the



Gray's System of Head Straps.

index column is shaken down) to these spots for a period at least of twenty minutes, and then the figures are read without removing the instruments. When a spot with increased temperature is found, the other thermometers are to be grouped about the suspected locality. Repeated tests show more or less sameness in the readings, so that it is possible to determine that a very limited portion of the brain is the seat of morbid action. In one case Gray was enabled to diagnose a tumor before death.

<sup>1</sup> Progrés Medical, 1877, quoted by Gray.

<sup>2</sup> N. Y. Med. Journal, August, 1878, p. 131.

<sup>3</sup> Revista Sperimentale di Freniatria e di Medicina Legale.

<sup>4</sup> The adjustment of these straps should be made so that those passing over the head should go in front and behind the fissure of Rolando which divides the important motor tracts. Gray measures from the fronto-nasal fissure, and fixes the location of the fissure as  $6\frac{7}{8}$  inches posterior to this point.



<sup>1</sup>Dr. Gray thus details the observations he made:—

"The patient was a female, aged thirty-four. There was present a typical 'choked disk,' marked pain in the temple and brow, becoming unbearable in paroxysms, nausea, vomiting, ptosis, paralysis of the ocular muscle. The first paroxysm of pain came on January 21st. The bodily temperature ranged near the normal. Upon these symptoms a diagnosis of intra-cranial tumor was made, probably situated at the base. Placing my thermometers upon the head, I ascertained the temperature at the different stations to be as follows:

|                      | Left.  | Right.  |
|----------------------|--------|---------|
| Frontal, . . . . .   | 96.75° | 98.33°  |
| Parietal, . . . . .  | 95°    | 99.75°  |
| Occipital, . . . . . | 96.75° | 100.50° |

The average of the two sides, if calculated, will be found to be 96.16° on the left, on the right 99.52°, the average for the whole head being 97.84°.

The rise above the normal averages is startlingly apparent. At the Left Frontal Station it was 2.39°; at the Left Parietal, 56.0°; at the Left Occipital, 4.09°; at the Right Frontal, 5.12°; at the Right Parietal, 6.16°; at the Right Occipital, 8.56°; while the average of the left-side had mounted above the normal 2.33°, the right side 6.66, and the average of the whole head 4.33°!

This particular observation was taken as I was at the outset of my study of the subject, and was made with my first set of thermometers, which, as I have already stated, were defective. I have satisfied myself, however, that the defect amounted to but a little over one degree. If, therefore, from these figures one and a half degree be deducted, all fear of error may be dismissed; and yet the increase is unmistakable. About this date (March 4th), I wrote Dr. Rockwell: "I shall certainly expect to see inflammatory changes from the base of the fissure of Sylvius backward along the occipital lobe, as well as that these changes shall be spread around the base of the fissure." The patient died March 16th. \*

\* \* \* \* \* "The meninges were found apparently normal, with the exception of a slight congestion. At the base of the brain the membranes and skull were to all appearances healthy. But a soft, jelly-like tumor, the size of a hazel-nut, was found between the horizontal or posterior branch of the fissure of Sylvius and the first temporal fissure, while the whole of the right occipital lobe was converted into a colloid, extremely vascular mass, which gave way under examination, this degeneration also extending anteriorly to the tumor as far as the fissure of Sylvius. There was no apparent disease except at these points. Upon microscopical examination, I ascertained the tumor to be a typical glioma, thickly strewn with small extravasations of blood."

Dr. Chas. K. Mills<sup>2</sup> has reported an interesting case of tumor of the

<sup>1</sup> Loc. cit.

<sup>2</sup> Phil. Med. Times, Jan. 18, 1879.

brain, involving portions of the first and second frontal convolutions, in which he found that the temperature obtained over the middle frontal station averaged  $1.50^{\circ}$  above that of the other stations.

The evidence collected by the few observers already mentioned shows the normal average temperature to be about as follows at the stations designated :

|                      | GRAY.          | MARAGLIANO AND<br>SEPPILLI. | BROCA. |
|----------------------|----------------|-----------------------------|--------|
| R. Frontal . . . .   | 93.71° . . . . | 97.07 . . . .               | 95.39  |
| L. " . . . .         | 94.36° . . . . | 97.16 . . . .               | 95.79  |
| R. Parietal . . . .  | 93.59 . . . .  | 97.07 . . . .               | 92.84  |
| L. " . . . .         | 94.44 . . . .  | 97.12 . . . .               | 91.49  |
| R. Occipital . . . . | 91.94 . . . .  | 96.71 . . . .               |        |
| L. " . . . .         | 92.66° . . . . | 96.81 . . . .               | 92.66  |

N. B. The experiments of Gray and Broca were made during cool weather.

Gray found the average temperature on the left side of the head to be  $93.83^{\circ}$ ; right,  $92.92^{\circ}$ . The average temperature of the whole head, exclusive of the vertex,  $93.51^{\circ}$ . Average temperature of motor region of vertex,  $91.67^{\circ}$ . His conclusions may be summed up as follows :

"If there be an alteration of temperature at any of the lateral stations of more than one and a half degree above or below the average temperature of such station, this fact will justify a *suspicion* of abnormal change at that point.

"If there be an alteration of temperature at any of the lateral stations of more than two degrees above or below the average of such station, this fact will constitute *strong evidence* of the existence at this station of abnormal change.

"In proportion as the alteration of temperature at any individual station is increased or decreased beyond the figures just mentioned, in exact proportion will the strength of the evidence be increased as to the existence of abnormal change at that station, until, the maximum or minimum having been passed, the evidence will become almost conclusive.

"Should it so happen that such elevation of temperature above the average should be at any lateral station on the right, causing a rise at this point beyond the average temperature at the corresponding station on the left, this would strengthen the suspicion or the evidence."

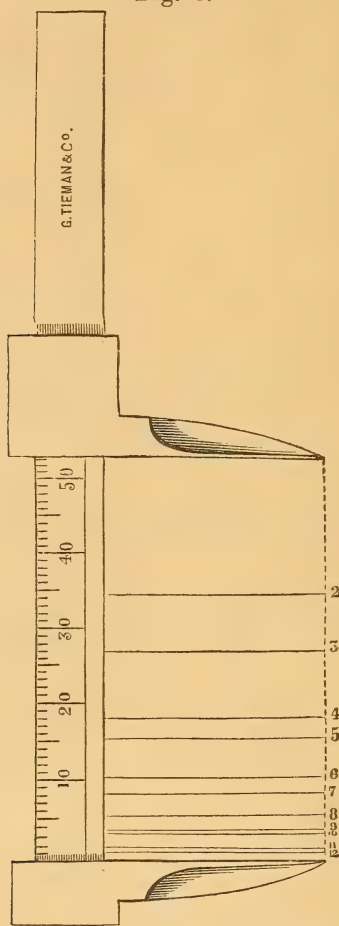
My own observations have been but few in number, though I trust I shall soon be able to add to Dr. Gray's valuable collection of facts.

In one case of undoubted cerebral tumor under my charge there is a rise of temperature of three degrees, which does not even vary a degree though I have made over thirty examinations under all sorts of circumstances. In one case of chronic cerebral meningitis, there was a general rise of cranial temperature, which was highest at the vertex, however.

The ÆSTHESIOMETER was first suggested by Sieveking, and has since been modified by different individuals. We have several different varie-

ties to choose from, but no one is better than the original instrument of Sieveking, which is also used and recommended by Brown-Séquard. It is made of brass or steel, and very closely resembles a shoemaker's mea-

Fig. 3.



Sieveking's Æsthesiometer.

sure. The movable slide and permanent arms at the end are sharp-pointed. The bar upon which the free slide moves is ruled in centimeters.

The other æsthesiometers are mostly shaped like dividers, and are open to the objection that the points are liable to be unconsciously approximated when the instrument is removed, so that the result of investigation is somewhat unreliable. Carrol's æsthesiometer has one advantage. The points are bifurcated, one arm ending in a bulb, while the other is sharp, so that analgesia as well as anæsthesia may be tested.

Dr. E. C. Seguin has made a very decided improvement upon the original instrument of Sieveking. He has had it constructed of aluminum, and of a smaller size, so that it is light and small, and may be easily carried in the pocket-case.

The principle upon which the æsthesiometer is constructed is the following: The normal receptivity of tactile impressions enables the subject to distinguish two points which are brought simultaneously in contact with the skin. This susceptibility varies greatly in different regions in proportion to the delicacy of the tactile sensation located therein. If there be loss of sensation as an accompaniment or result of nervous disease, of course the distance between them will have to be increased before the points will be felt as two. In hyperæsthesia they may be much more nearly approximated and distinguished as two than in the anæsthetic state.

The average distance at which the two points of the instrument can be felt in the normal state are as follows:—

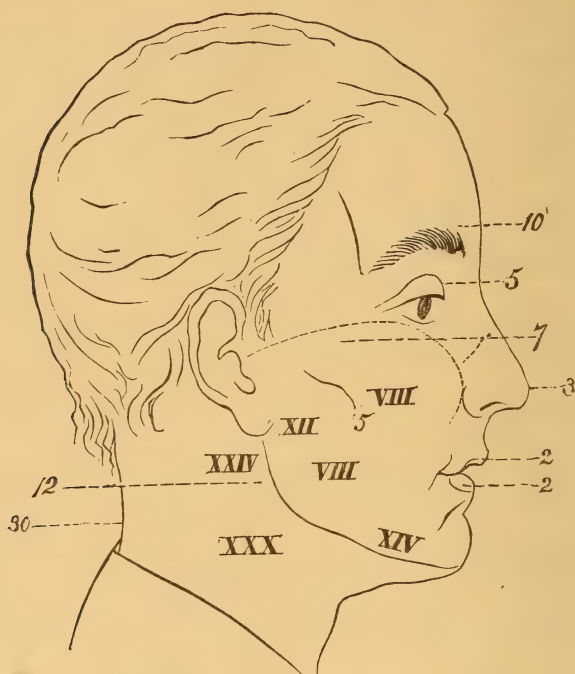
|   |                    |
|---|--------------------|
| Point of tongue . . . . .                               | $\frac{1}{2}$ line |
| Red surface of lips . . . . .                           | 2 lines            |
| Palmar surface of third finger . . . . .                | 1 line             |
| Tip of nose, . . . . .                                  | 3 lines            |
| Metacarpal bone of thumb . . . . .                      | 4 "                |
| Skin of cheek . . . . .                                 | 5 "                |
| Mucous membrane of hard palate . . . . .                | 6 "                |
| Dorsal surface of first finger . . . . .                | 7 "                |
| Dorsum of hand over heads of metacarpal bones . . . . . | 8 "                |
| Mucous membrane of gums . . . . .                       | 9 "                |
| Lower part of forehead . . . . .                        | 10 "               |
| Lower part of occiput. . . . .                          | 12 "               |
| Back of hand . . . . .                                  | 14 "               |
| Neck under lower jaw . . . . .                          | 15 "               |
| Vertex . . . . .  | 15 "               |
| Skin over the patella . . . . .                         | 16 "               |
| Skin over the sacrum . . . . .                          | 18 "               |
| Skin over the sternum . . . . .                         | 20 "               |
| Skin over cervical vertebræ . . . . .                   | 24 "               |
| Skin over middle of back . . . . .                      | 30 "               |
| Skin over middle of the arm . . . . .                   | 30 "               |
| Skin over middle of the leg . . . . .                   | 30 "               |

Certain precautions must be taken when using the æsthesiometer, or else our examination will be unsatisfactory in the extreme; we must not depend in all cases upon the patient's statement, but exercise tact in getting from him satisfactory answers, and not guesses. There seems to be in some individuals a discouraging stupidity which prompts them, in answer to the question, "How many points do you feel?" to oftentimes reply "Three," when they know that the instrument has but two points. It is of the greatest importance that the patient's eyes should be covered or that he should close them, as he will unconsciously look at the instrument during its application. It is also of moment that the points should be fairly and at the same time applied to the skin, one not being pressed



more than the other, and finally, it may be stated that they should not be applied at any place where the clothing has rubbed or chafed the surface.

Fig. 4.



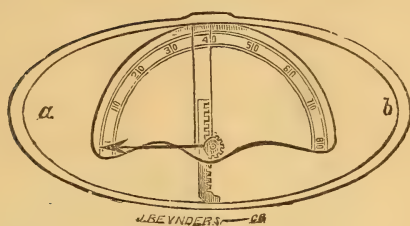
*Diagram for making Records.*—Roman numerals show anæsthetic indications, the others normal sensibility.

Since the appearance of the first edition of this book Dr. Hughes, of St. Louis, has devised a very convenient instrument, a new feature being an ingenious scale of measurements upon its bar, with a standard for reference.

Various tests of sensibility are simpler than those of the kind I have described. For gross tests the finger tips of the examiner may be applied and separated like compass arms. Shape and pressure may be determined by the application of various-sized bodies, weights, or coins, the subject's eyes being meanwhile bandaged.

**THE DYNAMOMETER.**—Various forms have been devised, that in general use being invented by Burq and introduced by Mathieu. It consists of an elliptical spring, which, when compressed in the hand, registers upon an index the force exerted. When the needle is forced ahead it remains at the point it had reached when pressure was remitted, and the spring expands. Its disadvantage lies in the inequality of pressure made at different times, the bulky character of the apparatus, and its inadaptability to other uses.

Fig. 5.



Mathieu's Dynamometer.

Having recognized the necessity for an instrument that would meet the therapeutical requirements not possessed by those of Mathieu or Duchenne, I have devised that figured in the appended illustration. It consists of a long glass tube (2) which dips into a small bottle filled with mercury. In connection with a bent brass pipe (3) is a rubber tube which terminates in a closed rubber bulb (5). When this bulb is compressed the mercury is forced up in the glass tube, the end of which is closed. Attached to the tube is a scale (1) registered on one side in pounds, and on the other by marks separated by regular intervals for the purpose of making comparative estimates. As fifteen pounds' pressure to the square inch is required to compress a given body of air into one-half its original space, of course a force of fifteen pounds' pressure brought to bear upon the bulb would be required to press the column of mercury half way up the scale. The advantages of this apparatus are the following:—

1. Its simplicity.
2. The adaptability of the rubber bulb to receive pressure exerted by all the flexors of the hand. Mathieu's spring is only acted upon by a limited number; at the same time, therefore, the test is not a true one.
3. The action of the muscles is the same at different times. The same group of muscles always being brought into play, accurate comparative tests may be made from day to day.
4. The part receiving the pressure is of a convenient shape to be used by persons with either small or large hands.
5. It is accurate and always gives reliable indication of the pressure brought to bear.

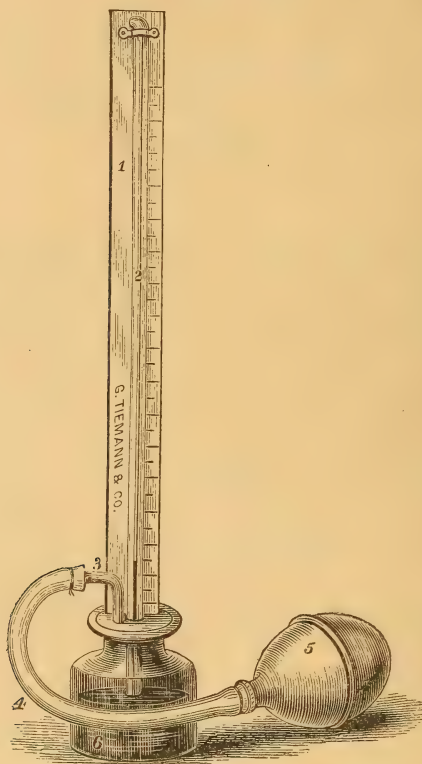
Dr. Birdsall of this city has recently invented a most ingenious foot dynamometer for testing the strength of the lower extremities.

The dynamometer is at best an instrument of questionable value, as are others requiring an effort upon the part of the patient. In rough tests of power it is useful, but in accurate case-taking, very little importance can be attached to the detailing of small variations as recorded upon the dial or scale of any dynamometer.

I have combined the rubber bulb with the drum of Maréy, and am enabled to obtain gross variations with tolerable accuracy. The drum has

two pipes, one of which is connected with the rubber bulb, while another is attached to the lower end of an open glass tube. The bulb-drum cavity

Fig. 6.



The Author's Dynamometer.

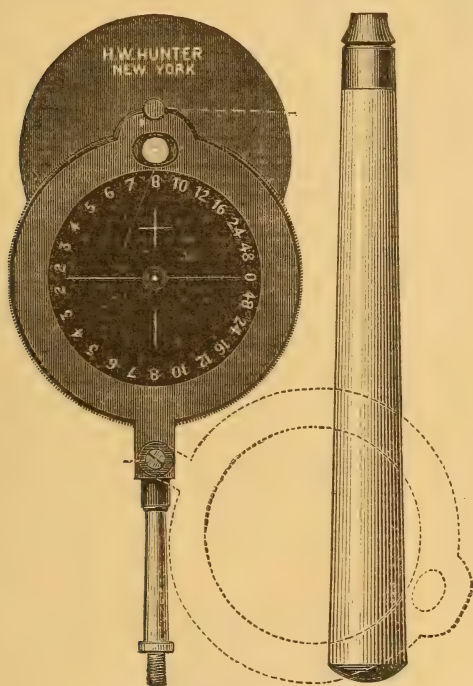
and a part of the tube are filled with colored fluid, so that the fluid in the latter reaches a mark at about the middle of its length. The patient grasps the bulb and makes enough pressure to force the fluid in this tube to a mark slightly above the other. The sustained voluntary effort required to keep the fluid at this point necessitates some delicacy of muscular co-ordination, and should this be impaired there will be expansion of the drum-head and consequently irregular tracings upon the cylinder of the registering apparatus. This cylinder should be covered by a piece of smoked paper, and the stylet placed in apposition thereto.

In alcoholic tremor, commencing sclerosis, and the metallic tremors, we may obtain very beautiful tracings.

**THE OPHTHALMOSCOPE:**—The parts composing the ordinary ophthalmoscope are the following: A concave mirror perforated at its centre, a series of lenses by which the refraction in the subject's or observer's eye

may be corrected, and a bi-convex lens. The three forms in common use are those of Liebreich, Loring, and Knapp. The two latter are essentially alike in construction, and the first is quite primitive, usually of bad construction, and quite unreliable.

Fig. 7.



Loring's Ophthalmoscope.

In the examination with this instrument great care should be taken by the observer to determine whether he or his subject possesses errors of refraction, and if so, to correct them with the proper lenses. In the modern ophthalmoscope a number of lenses are held in a revolving disk behind the mirror.

For more specific directions the reader is referred to Dr. Loring's admirable little work.<sup>1</sup>

To examine the eyes of a patient properly, the observer may follow the concise directions laid down by Hutchinson.<sup>2</sup>

"Having placed the patient's head in such a manner that the light (a lamp, candle, or gas-light) is on a level with his temple, and slightly be-

<sup>1</sup> Determination of Errors of Refraction with the Ophthalmoscope. E. G. Loring. Wm. Wood & Co., N. Y.

<sup>2</sup> Jonathan Hutchinson. Clinical Reports of London Hospital, 1867—8, p. 182.



hind it, and his face, as a consequence, in shadow, the observer sits in front and applies the ophthalmoscope mirror to his own eye. He should keep both eyes open that he may see where the light falls, and then move the mirror until the light falls full on the pupil of his patient. In a moment he will perceive the first fact which this instrument reveals, that the fundus is not black, as it has always appeared to be before, but that it is of a brilliant fire-red. He will, however, see nothing of the fundus distinctly, only a general red reflex. Now at this point the student must stop awhile and use his mirror, to inspect, first, the transparency of the cornea, and, next, that of the lens and vitreous, and to do this he must make the patient move his eye in various directions. After a little practice he will be able to manage his light well, and to throw it with precision wherever he may wish, and to keep it steadily on any given part. At a first lesson he may even, with advantage, practise for a while by illuminating the second button of the patient's waistcoat. Tact in directing the light having been obtained, we may now proceed further. Instruct the patient to look, not full in your face, but over one shoulder; if you are inspecting his *right* eye, over your *left* shoulder. You will, when he does this, notice at once that the tint of the light reflected from his fundus is changed, that it is no longer fire-red, but canary yellow. The reason of this is that a different part of the fundus is exposed to view, that, namely, of the optic disk itself, which is much lighter in color than the rest. The area of yellow is very large—occupies, indeed, the whole of the field, while we know that the disk itself is very small. This proves that the objects thus indistinctly seen are immensely magnified. Magnified by what? By the patient's own eye, which, as we have said, is equivalent to a lens of one inch focus.

“Hitherto we have seen nothing distinctly, but if the observer now brings his head very close to his patient's face, he will be able with more or less facility to observe the details at the bottom of the eye, the trunks of vessels of the retina, the optic disk, etc., etc. All these will be seen very large indeed, being still magnified by the patient's eye. What he sees now is equivalent to type looked at through a one-inch lens, placed exactly one inch in front of it.”

Without entering into an extended discussion as to the value of this

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NOTE.—Dr. Loring says, in concluding an admirable paper: “By the experiments considered in the foregoing remarks two alternatives are forcibly presented to our mind: either that the circulation of the eye is not a reflex of the circulation of the brain, though derived directly from it; and thus agents which affect profoundly the one have little or no influence on the other; or, if the retinal circulation is a reflex of the cerebral, it follows that the influence exerted on the circulation of the brain by agents at our command, remedial or otherwise, is very much less than heretofore supposed.

“I cannot but think that the former alternative is the more rational, and from that very independence of the two circulations there is reason to fear, so far as functional, and especially mental diseases, are concerned, that there never will be, any more than there now is, any art to read the mind's construction in the eye.”

instrument as a means of diagnosis, it will be well to state frankly that I do not believe that it possesses any positive value in the diagnosis of brain disease, *except where the condition of the fundus is the result of an organic disease of the brain or cord*, or when it is possible to connect such disorders with errors in refraction.

In making this statement I shall, perhaps, find many opponents, but I nevertheless have many powerful allies.

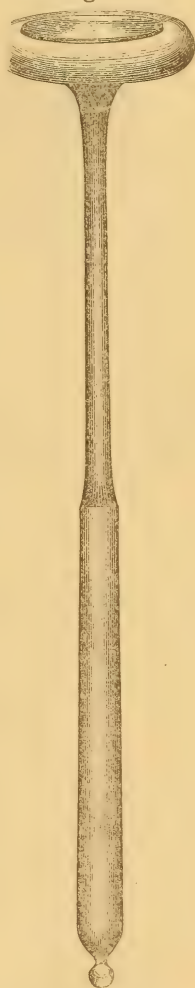
Bouchut,<sup>1</sup> Panas,<sup>2</sup> Albutt,<sup>3</sup> Bull, and others have written extensively, and have furnished a large number of clinical reports of ophthalmoscopic changes co-existent with cerebral tumors, meningitis, softening, effusion, cerebral hemorrhage, general paralysis, locomotor ataxia, and other forms of sclerosis, epilepsy, and the syphilitic and uræmic neuroses. Hutchinson,<sup>4</sup> of Philadelphia, in an admirable article, gives many of these cases, and shows the real value of the ophthalmoscope, especially when an examination of the fundus reveals choked disk and optic neuritis, but I will speak more fully in regard to this subject when we come to the discussion of special diseases.

My friend Dr. Buzzard, of London, demonstrated to me at the National Hospital for the Epileptic and Paralyzed, a useful application of the ophthalmoscope, for the purpose of testing the sensibility of the iris. The patient sits in a dimly-lighted room and looks at some object at a distance, so that the pupil is not contracted in accommodation. A pencil of light is then thrown upon the eye-ball to one side of the pupil, and gradually changed in direction, so that the iris is suddenly stimulated. Erb prefers for this test the use of artificial light concentrated by a convex lens.

**THE PERCUSSION HAMMER**—For the purpose of rapping the patellar or other tendons, the ordinary percussion hammer with a rubber head, such as is ordinarily used by medical men in chest examinations, has been adopted.

One with a flexible whalebone handle is the best. The patient seats himself with both feet upon the ground, with bared

Fig. 8.



Percussion Hammer.

<sup>1</sup> Du Diagnostic des Maladies du Systeme Nerveux par l'Ophthalmoscope. Paris, 1876.

<sup>2</sup> La France Médicale, Feb. 26, 1876.

<sup>3</sup> Med. Times and Gaz., vol. i., p. 495, and seq.

<sup>4</sup> Phil. Med. Times, May 8, 1875.

legs and a smart blow is then struck just below the patella, with the effect of producing the "tendon reflex" movement. A sharp contraction of the quadriceps femoris generally occurs in the healthy person, and a more or

Fig. 9.



Producing the "tendon reflex" movement. (Gower.)

less violent extension of the leg follows. This method of procedure may be resorted to, or the patient may cross his legs, and the examiner may tap the tendon of the dependent knee.

The position of the limb should never be constrained or uncomfortable, and there must be no voluntary contraction of the muscle upon the part of the patient. In cases where there is unusual excitability of the "tendon reflex" the blow may be struck upon the tibia. In fat persons the patient's leg may be supported upon the arm of the examiner, as figured in the annexed illustration. This subject will in another part of this work be alluded to more fully. (See "Diseases of the lateral columns, etc.")

## APPARATUS FOR THE TREATMENT OF NERVOUS DISEASE.

**ELECTRICAL.**—Two forms of apparatus are required—one for the production of *galvanic*, the other for the *induced or Faradic current*—as well as the necessary electrodes.

As we know, the galvanic current is derived directly from a battery or pile, the first consisting of two elements, which are contained in a vessel filled with some exciting solution, and the latter of plates of metal placed one above the other, and separated by disks of felt or paper moistened with a solution of salt or acid. This last apparatus is rarely used.

One vessel or cell of the form I have first described constitutes a *simple battery*, and two or more, with the poles alternately connected, a *compound battery*.

Two qualities of electric force are generated by a battery of this kind: 1. Quantity; 2. Intensity. The latter is the characteristic which makes it valuable as a means for the production of muscular contraction and nerve stimulation.

The Faradic current is derived from a galvanic cell primarily, and is developed by its passage through a coil of wire wound about a central core or bundle. Two currents are induced therein: one the *primary induced*, the other the *secondary induced*. The first is less coarse and violent in its effects than the other.

For a more extended description of electro-physics, physiology, and

therapeutics, I would refer the reader to any of the works mentioned at the foot of this page.<sup>1</sup>

For the production of the galvanic current, we may avail ourselves of either one of the permanent batteries; the cells of which may be set up in the cellar, and the wires carried to a proper board in the office, containing apparatus for their selection; or we may use the ordinary portable galvanic battery, many styles of which are made.

I have given the Leclanche battery a fair trial, and now do not recommend it, as it is dirty, inconstant, and rapidly loses power. The "magazine battery" of Chester, in which the peroxide of lead is substituted for the black oxide of manganese in the porous cell, is much better. The old Daniel's cell is, I am convinced, the best of all, and whether in the form of the Siemens and Halske, or Hill modification, is all that can be desired.

The table board of Fleming of Philadelphia, or the arrangement known as the "cabinet battery," which is made by the Galvano-Faradic Company of New York, is admirable for office use.

The Faradic instrument should be provided with an attachment for the slow or rapid interruption of the current, an addition to the ordinary battery, which will be found of immense advantage in certain forms of paralysis. The instruments of the two firms I have mentioned, besides those of Drescher and Kidder, are all good.

Two or three cotton-cloth covered electrodes of different sizes, or flat sponges with rubber backs, with fine wire pole cords instead of the flimsy gold-thread connections in present use, which oxidize and break, will be needed, as well as a bundle of fine wires held in a handle, which is known as an *electric brush*. Static electricity has lately received some attention. Beyond its moral effect upon the patient, especially if there be hysteria, I do not believe that it possesses any advantages over the chemical currents.

RUBBER MUSCLES, ETC.—Dr. Van Bibber, of Baltimore, has devised a very useful apparatus for the treatment, especially of lead paralysis. It consists of a strap for the hand or other part which needs support, and one for a point of attachment of the muscle. When properly

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Either of these works will be found practically useful to the student:—

Tibbit's Handbook of Medical Electricity.

Reynolds' Clinical Uses of Electricity.

Althaus's Electricity, Theoretical and Practical.

Poore: A Text Book of Electricity, etc.

Lincoln's Electro-Therapeutics.

Beard and Rockwell's Medical and Surgical Electricity.

Hamilton's Clinical Electro-Therapeutics.

Duchenne's de l'Electrisation localisé, 1872.

Onimus et Legros, Traité D'Electricité Med.

Benedikt Electrotherapie, 1874-5.

Ziemssen, Die Electricitat in der Med., 1872.

Besides, the works of Rosenthal, Erb, Meyer, Eulenburg, and others.



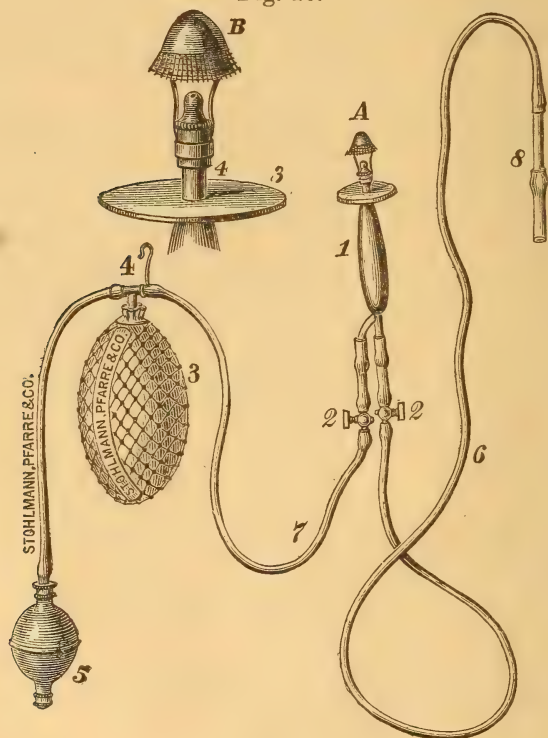
applied, the rubber pipe, which takes the place of the paralyzed muscle, raises the hand, so that the strain upon the enfeebled muscle is relieved. Dr. Van Bibber has also used court plaster for the treatment of ptosis and other minor paralysis.

THE HYPODERMIC SYRINGE, ETHER SPRAY APPARATUS, and SPINAL and CRANIAL ICE BAGS, should be procured by every physician who has occasion to treat this class of diseases.

CAUTERIES.—Until a few months ago the old forms of cautery were used almost exclusively. These are of iron, and are sometimes platina covered. When needed, they are heated in the flame of a Bunsen burner, Russian blast lamp, or some such contrivance, but lose their heat very rapidly, and generally assume a dead red color when applied. The glass rods, heated in a like manner, though somewhat more convenient, become very quickly cool.

Dr. J. J. Putnam, of Boston, exhibited at a meeting of the American

Fig. 10.



The Author's Gas Cautery.

Neurological Association the first gas cautery seen in this country, though Alex. Bruce years ago invented a cautery of this kind. It

was constructed in such a manner that the jet of an ordinary gas blow pipe was directed upon a cup of platinum. Its advantages over the older variety were manifold, but it possessed faults I have tried to remedy in a modification.

The advantages of this instrument are the following:—

1. The jet which prevents all hissing or noise, and still produces a very powerful blast.

2. The apron of wire gauze, which prevents the return of flame, thus obviating the danger of burning parts that we do not wish to affect.

3. The large bag, which acts as a reservoir, so that the operator need not use the rubber bulb nor watch the burner after it is filled.

4. The hook, which enables him to suspend the bag and tubing from his person, thus removing all drag.

The general advantages of this form of cautery are important. A uniform heat may be kept up for hours with very little exertion. The furnace, which is not only inconvenient, dirty, and alarming to timid people, but is a slow method, is done away with. In less than a minute the platinum dome can be heated to whiteness.

The cauteries of Pacquelin and Guérard, of Paris, are both good. In them the vapor of benzine (which should be impure) is forced with air upon a piece of hot platinum. These are excellent substitutes for the cautery I have just described, especially in the country, where there is no gas. Messrs. Stohlman, Pfarre & Co. have constructed for me an apparatus which consists of the cautery, handle, and a hard rubber receptacle containing charpie which is to be saturated with benzine. There is no danger of explosions such as exist when we use the ordinary bottle that forms a part of the French instrument.

It has been recommended that the spinal ether spray be used to deaden pain; but not only is there danger of an explosion when this procedure is tried, but it seems to me that the very object of the operation, *revulsion*, is not accomplished, as the peripheral filaments are of necessity benumbed.

## CHAPTER I.

## DISEASES OF THE CEREBRAL MENINGES.

ALL of the investing membranes of the brain may be the seat of inflammatory action, but it is almost impossible in certain instances to make distinctions between inflammation of the arachnoid and pia mater, though this has been attempted by Parent-Duchatelet,ALLEMAND, and others. We will, therefore, have to content ourselves with a division founded upon the duration, intensity, and coexisting diseases of the general system, and limit our regional diagnoses to forms which may be called meningitis of the convexity and meningitis of the base.

In respect to certain circumstances which modify the appearance of the disease we may divide its varieties as follows:—

|  |   |
|--|---|
| Cerebral pachymeningitis,<br>(Inflammation of the dura mater,) | { Acute,<br>Chronic,<br>Chronic, with hæmatoma. |
| Acute cerebral meningitis,                                     | { Basilar,<br>Of the convexity,<br>Granular.    |
| Chronic cerebral meningitis.                                   |   |

## PACHYMENINGITIS (INFLAMMATION OF THE DURA).

Two forms of pachymeningitis are to be met with, one of which is acute and is the direct result of injury or disease of the cranial bones, and is generally fatal in a short time; and the other, of a chronic nature, which may either remain after injury, or arise from some intracranial cause, or perhaps be the result of general disease, or old age.

## ACUTE PACHYMENINGITIS.

**Symptoms.**—After the traumatism, or when the external disease has invaded the intracranial cavity, the first symptom is usually severe and localized pain, which finally extends with the inflammation, and becomes diffused over the entire head.

Rigors, alternating with elevation of temperature, which may sometimes attain 105° or 106° F., head pain and occasionally spasms of the arms or legs, are ordinary symptoms; and if the condition be a very acute one, there may be general convulsions, or perhaps a partial paralysis, which is unilateral.

Delirium usually supervenes in from three days to a week, and coma ends the disease, should an effusion of blood take place, and this is a common termination.

The pulse during the first two or three days varies from 60 to 70, while towards the end it becomes much more frequent and very full. During the invasion, and after the disease is fully established, especially if the inflammation extends to the base, the head may be drawn backwards and downwards.

Ramskill<sup>1</sup> has called attention to the hyper-sensitiveness of the cornea, and I have been often impressed by another symptom, viz., the redness of the conjunctiva and the constant tendency to lachrymation. Vomiting very commonly takes place, and is always quite a suggestive symptom of meningeal trouble. When the disease follows otitis its onset is not so sudden as when it is the result of injury, but a train of symptoms of gradual appearance marks the extension of the morbid process step by step, though in some instances rigor with sudden coma may be the first indication of mischief. This is in most cases the purulent form. Cases of the idiopathic variety of pachymeningitis are quite rare, although several have been reported by Abercrombie and other older writers. One case related by the former authority may be worth mentioning. This writer also gives six others which originated from middle ear disease or abscesses in other bony cavities. These latter cases are not uncommon, if we may accept the experience of aurists and surgeons. Abercrombie's<sup>2</sup> patient, in whom the disease was idiopathic, died in fifteen days. The first indication was severe pain in the left temple, which continued for two weeks, when a "swelling" appeared beneath the left upper eyelid. Four days before her death violent convulsions took place, which were preceded by slight rigors. The swelling was punctured, and a considerable quantity of pus escaped. A probe passed into the opening came in contact with bone, and could be inserted for some distance, the end being in contact with the roof of the orbit. During previous days her condition had varied to a great degree, and at times she seemed to be very comfortable. On the day before her death she complained of vertical headache, became semi-comatose, and died in this state. Extensive discoloration, thickening, and other changes in the dura mater were found with adventitious membrane and pus. In a case detailed to me by Drs. White and Asch of this city, there was alternating paralysis associated with aural disease which affected the ears in turn.

Fizeau<sup>3</sup> mentions a case which closely resembled this one, and another quoted by Abercrombie, and seen by Pratheron, was also of idiopathic origin. Abercrombie's other cases presented common symptoms which were traced to assignable causes. Dr. Clark<sup>4</sup> has presented five cases of the

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<sup>1</sup> Russell Reynolds' System of Medicine, vol. ii., page 325.

<sup>2</sup> Abercrombie on the Brain, page 21.

<sup>3</sup> Journal de Médecine, tom. ii., New Series, page 523.

<sup>4</sup> Transactions New York Pathological Society, 1876.



acute form, due to otitis. Dr. Bauduy another which followed scarlet fever, and many of the same kind may be found mentioned by other authorities.

#### CHRONIC PACHYMEINGITIS.

A far more interesting class of cases are those which have lasted for some time, and have invaded the underlying membranes, ending in involvement of the cortex cerebri. The following is a fair example:—

**Symptoms.**—John McM., age 30, of temperate habits. The patient was a young man of the laboring class, and was employed in a machine-shop at the time of the accident. Three years ago, while turning a piece of metal, it caught upon the end of his turning tool and flew out of the lathe (which was driven by steam-power), striking his head, and cutting a scalp wound over the upper part of the right parietal bone. He fell unconscious, and was carried to his home, remaining in the same state for about eight hours. After this he recovered slowly, was delirious, and evidently had had convulsions. From this period to the time when I saw him his history was not very clear, but he had had convulsive paroxysms from time to time, and severe headache, which he complained of when he came for advice. This pain was limited to the right side of the head, and principally centered at the injured spot. His face was quite puffed and swollen, and his eyes were red and watery. Pressure upon the cicatrix caused intense pain. His right pupil was slightly enlarged, and he complained that his vision was imperfect. Sleep was disturbed by the pain which would often occur in paroxysms of a very intense character. He complained that his left arm felt stiff, and that his fingers were cold, but I was unable to find any loss of power. He continued in this state for a year or more, and when I next saw him his speech had become slow and hesitating, and his face wore rather a silly expression. He then complained of some feebleness of the left arm and leg. The headache had not abated, and the convulsions had been much more frequent. His friend who came with him stated that his mind had greatly changed, that his behavior was eccentric, and that he had had delusions of various kinds. I subsequently lost sight of him. In some features this case resembles one of softening. This form of chronic pachymeningitis is much more obscure when it is connected with syphilis. There is not only a great disproportion between the severity of the symptoms and the extent of the morbid process, but symptoms of great variety may be evinced as expressions of pachymeningitis of syphilitic origin.<sup>1</sup> Lagneau fils<sup>2</sup> reports a case in which the only symptom was headache, which was most violent at night. *Post-mortem* examination revealed pachymeningitis over the anterior lobes of the cerebrum, with bony plates and some sclerosis of the brain-substance. There was, in addition, extensive perforation of the ethmoid bone. Instances are related by Gama where the patients had died conscious,

<sup>1</sup> Trans. N. Y. Path. Soc., vol. i., p. 13.

<sup>2</sup> Observation 3, Lagneau, *Maladies syphilitiques du Système nerveux*. Paris, 1860.

and their meninges were found to be decidedly affected. Keyes,<sup>1</sup> in a most complete and exhaustive memoir, presents a number of cases of hemiplegia which were the ultimate result of the meningeal inflammation, and calls attention to the pain which precedes the hemiplegia, and which is always produced when pressure is made upon the cranium. A feature of the hemiplegia is the absence of any loss of consciousness.

Syphilitic meningitis of this description is very often—I may say almost always—symptomized by a decided failure in the mental powers, which begins in fact as soon as the pathological process manifests itself by any symptoms at all. I regard this slowness of intellectual action which, by the way is general, as almost pathognomonic. In some cases it has been almost the only symptom of a pachymeningitis which was not recognized until after death. I have, since the appearance of the first edition of this book, been called to see several persons, who have subsequently died, presenting an imperfect hemiplegia—that is to say, a hemiplegia of a comparatively light character, but associated with an equally light coma, lasting several days. There was not even laborious breathing, and it was possible to rouse the patients. It strikes me that in such cases the pressure had been quite gradually developed, and the cerebral mass had become to a degree accustomed to the pressure of the new deposit. <sup>2</sup>Bumstead and Taylor thus describe the later stages of syphilitic meningitis: “A general adynamic condition sometimes supervenes in patients affected with chronic inflammation of the meninges, which either ends fatally or renders them hopelessly bedridden. This weakness may be due to mere lack of innervation, or may be complicated by mild ataxic phenomena, characterized by unsteady gait and uncertain movements. The dullness of intellect by day is succeeded by nocturnal delirium. When lying in bed such a patient resembles one in typhoid fever, but there are marked points of difference. He is sleepy and dull, and his face is utterly expressionless. The tip and edges of his tongue are red, but the organ is never, unless late in fatal cases, dry, cracked and covered with sordes. Anorexia and constipation are often quite marked. The pulse ranges from 80 to 110, is full and not wiry. The temperature may be elevated in the morning to 100° F., and at night to 103° or 104° F. If conscious, the patient complains of intense headache and weariness. In a week or ten days he passes into a condition of complete unconsciousness, perhaps broken by brief lucid intervals. The urine and feces are passed involuntarily. If not relieved, the condition soon becomes more serious; the temperature continues to rise, and the pulse increases in rapidity; no food is taken, and the stupor merges into fatal coma.” The above account is a most graphic one, and is a striking picture of a common form of trouble.

Fournier is inclined to fix the time for the development of syphilitic

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<sup>1</sup> Syphilis of the Nervous System. New York, 1870.

<sup>2</sup> The Pathology and Treatment of Venereal Diseases by Bumstead and Taylor, 4th edition, p. 655.

meningeal symptoms much later than those authors who have met with these symptoms in quite recent cases.

Of my own cases I have never seen syphilitic pachymeningitis before the end of the third year, and in most instances at least six or eight years after primary infection. In the case seen with Dr. Asch the development of symptoms followed at least fifteen years after the primary disease. It is probable, however, that there are cases of acute trouble with early development of active meningeal inflammation.

A form of syphilitic pachymeningitis may follow external syphilitic disease of the cranial bones. I may illustrate the features of such an attack by the following case, reported by Dr. Jas. R. Wood:—

Marie C., aged 20, was admitted to Bellevue Hospital, on account of an eruption of two weeks' duration, which had steadily progressed from a few points until it had become general, being most profuse on the face, neck, arms, and scalp.

The eruption presented a distinct coppery hue, and was of two varieties. There were three rupitic phlegma on the head, each of which contained a little pus, and three or four on the shoulders and back of the same character. The rest were tubercular.

She stated that, though often exposed, she had never suffered from primary syphilis, but that there was a sore on her thigh, near the vulva, which appeared two weeks before the eruption.

On examination, a simple chancre was found at the point complained of; there was also a chancre of limited extent in the vagina. Soon after admission she was observed to have a shuffling gait, and when questioned about it stated that her right arm and leg "seemed to be getting weak." The treatment consisted in the use of the corrosive chloride of mercury in Huxham's tincture of bark, combined with generous diet.

The eruption on the scalp was left undisturbed. The quantity of pus contained in each point was quite small, and it was deemed best to let them alone. One of them situated over the parietal bone of the left side was something larger than its fellows; none of them, however, increased in size materially.

There was very little improvement in the eruption, but the hemiplegia steadily increased.

Her appetite became poor, she began to have vomiting, and exhibited a cachectic appearance. The bichloride was necessarily discontinued, and mercurial vaporization substituted.

The hemiplegia became more complete, and her mind began to be obscured. The stupidity gradually deepened into profound coma, in which condition she died on the 30th.

*Autopsy.*—There was a denudation of the parietal bone of the left side of the periosteum, at a point corresponding with the rupitic spot above spoken of.

On removing the calvarium, the dura mater was found inflamed and firmly adherent to the skull, just beneath the denuded spot on the parietal bone and the eruption.

A small opening was found communicating between them, perforating the cranial walls, and looking very much like a worm-hole.

The brain at a point corresponding with the inflamed dura mater presented a greenish appearance.



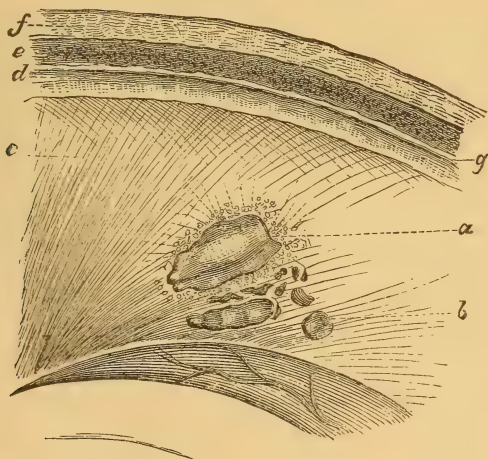
There was also an evident fulness and fluctuation. On making an incision an abscess was discovered which contained about  $\frac{3}{4}$  of pus. The other organs were healthy.

As a result of continued congestion we may have a form of pachymeningitis such as follows chronic mania. I have seen this change repeatedly as a secondary condition, but it must be confessed that the other meninges were as well affected.

**Causes.**—They may be briefly enumerated as external injury, otitis, syphilis, alcoholism, and various acute diseases, among them rheumatism.

**Morbid Anatomy and Pathology.**—In the majority of cases the inflammation is transmitted to one or more of the important sinuses. The most favorable points for the extension of disease of the temporal bone are the narrow space between the mastoid cells of this bone and the transverse sinus, and that between the cavity of the tympanum and the jugular fossa; and the proximity of the auditory meatus to the petrosal sinus, and the different canals which contain the nerves, to adjacent intracranial parts. The bony walls between these locations are of a perforated and lamellar character, and when attacked by caries are very apt to be destroyed.

Fig. 11.



*Osteoma of Dura Mater (Lancereaux).*—a. Bony Plate. b. Perforation. c. Falx. d. Dura Mater. e. Parietal Bone. f. Scalp.

If the disease be of a syphilitic nature there is generally a gummatous deposit scattered through the tissues, and the under surface of the dura mater is often covered by a syphilitic exudation which can rarely be mistaken. If it be the result of a traumatism, the membrane is seen to be thickened, opalescent, and congested. In old cases it is found to be closely adherent to the cranial bones, or it may contain long plates.

In this form of inflammation the morbid changes may be seen best at the convexity.



**Prognosis.**—The outlook is invariably bad, for in one variety the patient is carried off in a few days, or, should the disease become chronic, its progressive nature must lead us to expect an ultimate implication of other parts, and cortical softening or sclerosis and atrophy are probable terminations.

**Treatment.**—Treatment should be directed in the beginning to the cause, and if there be otitis, a free escape of pus should be provided for, and counter-irritants, topical applications, and leeches should be employed. If the pachymeningitis be attended by much pain, cold to the head and free administration of the bromides will be of service. The leeches may be applied to the tragus of the ear, or to the mucous membrane of the nostril.

#### CHRONIC PACHYMENINGITIS WITH HÆMATOMA.

It has been the custom, among certain writers lately, to speak of hæmatoma as an inevitable result of pachymeningitis. This, I think, is a mistake, for the production of blood-cysts is not the invariable rule. If, however, the thickening of the dura mater is excessive, there may be a gradual destructive process, which will be described when we come to speak of the morbid anatomy and pathology of the affection.

The disease may begin as I have already described, and may advance to a certain point before the grave symptoms which indicate rupture and consequent meningeal hemorrhage are expressed. These may vary in intensity in proportion to the extent of the effusion, which may be even so great as to produce sudden death, but such an early result is exceptional. The course of the disease is generally more gradual, and there is at first an initial hemorrhage of slight extent, which is followed in a great number of cases by two or three others. In some respects this effusion resembles cerebral hemorrhage in the production of acute symptoms, but they are nearly always less profound; and it is not so frequently followed by complete paralysis.

**Symptoms.**—The early symptoms of pachymeningitis that I have enumerated are those preceding the immediate evidences of the effusion. They may be reinforced by loss of memory and stupidity, and after a few months there may be a transitory loss of consciousness and incomplete hemiplegia which is characterized by much hyperæsthesia.

The phenomena of the attack are thus described by Huguenin:<sup>1</sup> “Severe headache, just before the attack; after loss of consciousness has occurred, contracted pupils, not reacting; in a few cases, paralysis of the facial nerve, on the side opposite to that of the hæmatoma; sometimes hemiplegia. These latter symptoms only occur in one-sided hemorrhages. A marked change in the color of the face is another of the symptoms reported. At the commencement of the attack, which is usually sudden, the face becomes flushed; the pulse is full and rapid, but soon grows small

<sup>1</sup> Ziemssen, *Cyclopædia of the Pract. of Med.*, translation, vol. xii., p. 409.

and irregular, and pallor succeeds the flushing. In some cases the pulse is slow; in others there is an increase in rapidity, continuing up to the time of death. Contractures of the extremities, and slight transitory twitchings, were present in a few cases."

Instead of hemiplegia there may be one-sided convulsions, but these depend very much on the degree of pressure exerted upon the cortex-cerebri. The condition, strange to say, is sometimes arrested after an indefinite period, and there is a return to the normal state, but traumatic hæmatoma is usually fatal.

Schuhberg<sup>1</sup> assents to the view held by Herschl, Virchow, and Cruveilhier, that hæmatoma is always the result of fibrinous inflammation, and believes that the prognosis is grave. In this paper he considers the duration of a fatal case to be about one month.

**Causes.**—Hæmatoma is a disease of adult life, and twenty-two per cent. of the cases collected by Huguenin were between the seventieth and eightieth years, and Durand-Fardel found that 77.4 per cent. of all cases were men, and 22.6 per cent. were women. As causes may be mentioned various cachectic and other diseases, among them Bright's disease, scurvy, syphilis, typhus fever, rheumatism, smallpox and scarlatina, alcoholism and sunstroke, or any condition which is conducive to continued hyperæmia of the dura mater.

**Morbid Anatomy and Pathology.**—The process involved in the production of hæmatoma is an exceedingly complicated one, consisting in the production of new vessels and new layers of fibrine due to the extravasation of blood. The first layer of this new tissue-formation takes place in contact with the arachnoid, and ultimately others form and become organized. The formation of the blood-cyst is due to the rupture of one of the new vessels, and the extravasation becomes surrounded by a layer of tissue which may be so firm as to preserve the cyst contents unchanged. This is particularly the case in the smaller cysts. The skull is sometimes found to be thin as seen by Hyrtl,<sup>2</sup> but this is not common, and some writers, among them Textor<sup>3</sup> and Rokitsansky,<sup>4</sup> consider that the reverse is to be seen in a greater number of cases. I may briefly enumerate the *post-mortem* appearances as follows: Beneath the dura mater may be found a layer of coagulum which contains fibrinous shreds binding it to the membrane itself. If the case be of long duration several layers of false membrane containing bloodvessels are to be found attached to the dura, and the late formations may be distinguished from those of early origin. Between these layers it is not unusual to find the results of interstitial hemorrhages which exist as blood-clots in different styles of organization. The thickening of the dura mater is thus described by Fox: "In the non-purulent form of the new formation, the result of inflammation be-

<sup>1</sup> Schmidt's Jahresbericht, vol. 104, pp. 164, 165.

<sup>2</sup> Ziemssen's Encycl., vol. xii. Am. Tran., Art. "Meningitis."

<sup>3</sup> Würzburg Verhandlung, vii. 1857.

<sup>4</sup> Rokitsansky, quoted by Huguenin.

comes very quickly the seat of vessels and is composed of several layers; those nearest the dura mater being composed of compact lustrous connective tissue fibres almost as dense as the dura mater itself, whilst the layer further removed from the dura mater is rich in cells with small narrow vessels, and the layer nearest the arachnoid, often firmly uniting the arachnoid to the dura mater, is remarkable for very large capillaries."

The size of the hæmatoma may vary from that of a small bean to that of an orange, and in one case, the autopsy of which was made by Dr. Huber of the Colored Home, the blood-cyst covered one entire side of the brain, and was fully an inch in depth. The patient was under the care of Dr. Whitall, who kindly contributes the following notes:—

P. B., 60, widower, N. Y.; mulatto; father, mother, and one brother died of phthisis. The patient has been intemperate, but now drinks only in moderation. He denies venereal disease; twenty-five years ago he had smallpox, and has since had intermittent fever and cholera. His trouble dated from an injury seven years ago. He was thrown from a hay-truck to the ground, falling upon his head, and causing blood to flow from his left ear; but he was able to walk to his home, one mile distant. He seems to have received no very serious injury, if we may judge from the immediate symptoms. Since the fall he has been troubled with headache off and on, increased by approaching a fire. He cannot appreciate the ticking of a watch pressed to his left ear. About a fortnight ago he had a chill, fever, and cough, some pain in back, with soreness around the whole gluteal region. Urination was slow, disturbed, and at one time he was unable to pass water; at another it would be too free; has been growing weaker since.

*June 15, 1874.* On admission patient was confined to bed; owing to apparent weakness in lumbar region he was unable to stand. In a few days he began to improve under the administration of iodide of potash. Walks with a staggering gait, and cannot follow a straight line. On closure of eyes does not have a tendency to fall. Heavy expression of countenance. No diminution in acuteness of sensibility can be discovered over any portion of the body. Had incontinence of urine on admission; is not so troubled at present time. Can walk about the ward; at times can dress without assistance. To-day complains of frontal headache; sleeps very soundly, with stertorous breathing. Appetite good; bowels constipated.

*24th.* Staggering gait, and inability to walk in a straight line, still present. If he closes his eyes while standing, there is a tendency (which by an effort he can overcome) to fall backward. Complains of pain on right side of head and face; sleeps most of the day in a chair; at night snores loudly. Bowels constipated. Nocturnal incontinence of urine exists.

*Feb. 6, 1875.* To-day, while patient was sitting in a chair, he had a convulsion, and then became comatose. Urine albuminous. Ordered *ol. tiglli*  $\mathfrak{m}$  iv, after the action of which he appeared much better.

*15th.* Very little change in patient's general condition since above note. Is still apathetic, and complains of pain in the head, on right side especially. There is still right facial paralysis, with somewhat diminished sensibility in this region. The tongue deviates, if any, to the right. Pu-



pils normal in size and reaction. No notable change in hearing. No loss of motion, though the right arm and leg are weaker than the left. The lower limbs (left more readily than right) can be drawn upwards, and extended with little trouble. He is unable to walk or stand without being supported, as the right leg gives away; complains of a considerable pain in the upper portion of the limb. Has occasional involuntary passages of urine and feces; as a general thing, however, the bowels are confined; urine evacuated with considerable force.

*March 19.* Appears to be losing strength very rapidly. Will not answer when spoken to. Temp.  $99\frac{1}{4}^{\circ}$ .

*21st.* Died about 9 P. M. comatose.

*Autopsy 36 hours post-mortem*—Rigor mortis marked. Body slightly emaciated.

The dura mater was found very firmly adherent to the calvarium to the right of the longitudinal sinus, and over a considerable portion of the convexity. After removing the dura mater, the pia mater on the left side was discovered to be unusually dry and congested, with here and there slight patches of lymph. The convolutions throughout this hemisphere were greatly flattened, and the sulci nearly obliterated. In the right cranial cavity a large hæmatoma existed. The tumor pear-shaped, with a larger extremity anteriorly, extended from the anterior portion of the second frontal convolution to the posterior portion of the second temporal, and from within an inch of longitudinal fissure to junction of lateral portion with base of skull.

The right hemisphere was correspondingly compressed downwards, backwards, and to the left. The depression corresponded to the shape of the tumor and was so situated that the greatest amount of pressure came upon the left lateral ventricle. The dimensions of this growth were as follows:  $6\frac{1}{2}$  inches antero-posteriorly; 4 inches vertically in greatest diameter; and about two inches in thickness.

In addition to the hæmatoma, a serous cyst (about the size of a hickory-nut), evidently originating from an old hemorrhage in the subjacent brain structure, the cicatrice of which still remains, was seen beneath the anterior lobe. Back of this another cyst, the walls of which were chiefly composed of softened brain tissue, was discovered, which, upon closer investigation, was ascertained to be continuous with the right lateral ventricle through the middle cornua. The right ventricle was greatly distended by serum, while comparatively little could be detected in the left.

In the left ophthalmic artery a long, slender clot, partly dark and partly translucent and yellowish, existed. No thrombi were noticed in the slight atheromatous arteries at the base of the brain.

No connection existed between the pia mater and the hæmatoma; the relations between it and the dura mater were so intimate as to require dissection before a separation was possible.

The petrous portion of the right temporal bone was considerably larger than the left, and, upon section, proved to be much more porous. No other abnormalities were present; no evidence of fracture at the base.

The way in which the tumor, though situated on the right side of the brain, pressed upon the left ventricle, explained the symptoms which, during life pointed to an involvement of the left side; and also offered an explanation as to the manner in which the fluid was forced through the middle cornua of the right ventricle.



*Heart*.—Very flabby; cavities dilated, and filled with dark coagula. Aortic valves were slightly thickened, and the artery was atheromatous. Mitral valves thickened.

*Lungs*.—The right was firmly bound to chest; very soft and congested. The surface was studded with pigment.

The left had also become adherent to parietes, and, at the apex, a few softened, cheesy points were discovered.

*Spleen*.—Enlarged and congested.

*Liver*.—Normal.

*Kidney*.—Cortex somewhat thicker than usual; both organs were waxy.

*Weight of the organs*.—Heart, 10 oz.; spleen, 7 oz.; liver, 55 oz.; right lung, 29 oz.; left lung, 18 oz.; right kidney, 6 oz.; left kidney, 5 oz.

**Prognosis**.—The existence of a blood tumor of this kind is not always a serious matter. Even after two or three extravasations have occurred, a retrogressive course takes place; but this is rare. Griesinger<sup>1</sup> reports a case in which partial recovery has taken place; and in 1876 the patient was still alive, and presented slight evidences of his former serious trouble. This termination of the disease is exceptional, however.

**Treatment**.—What has been said in regard to the management of uncomplicated pachymeningitis is applicable in this disease; and, in addition, venesection has been advocated by more than one authority. It should be employed during the comatose stage which marks the occurrence of an effusion, and at the same time a drastic cathartic will be found to be of excellent service. High living and excessive use of tobacco and alcohol are to be interdicted, and iodide of potassium may be given with the idea of producing absorption of the new growth.

## ACUTE CEREBRAL MENINGITIS.

The term meningitis has been applied, clinically speaking, to that form of inflammation which involves chiefly the arachnoid and pia mater, and in its acute form may be expressed by the following grave and alarming symptoms:—

**Symptoms**.—These may be divided in regard to their appearance into three stages: 1st. The stage of excitement or irritation; 2d. The stage of delirium; 3d. The stage of stupor.

An hypothetical case may be presented. The patient complains of a slight *headache*, which increases toward the end of the first twenty-four hours. It may not be attended by much annoyance, and he is usually able to attend to his daily duties, but during the succeeding six or eight hours it may become greatly aggravated, and is attended by *restlessness*, *flushing* of the *cheeks*, throbbing of the temporal vessels, and general discomfort. After a few hours there may be slight rigors or a severe *chill*, which is often mistaken for ague; and the rapid *elevation of temperature*,

<sup>1</sup> Archiv. der Heilkunde, 1862.

and hard, bounding pulse may strengthen the suspicion. The headache continues, and is still not confined to any particular locality, but is so intense that the patient seeks his bed, where he may lie, moaning, sighing, or tossing restlessly to and fro. The muscles of the legs may twitch, and the least noise, such as the creaking of a door, invariably irritates and startles the invalid; bright lights distress him, and he closes his eyes instinctively. He keeps his hands over his ears so that he may not hear noises in the room, or firmly presses his aching temples. There may be *vomiting* which is not dependent upon the condition of the stomach, is not attended by retching, and occurs whether the stomach be empty or full. If the patient be a child, there are generally *convulsions* of a very violent character. These constitute the first stage.

*Active delirium* usually appears during the first two days, and continues through the greater part of the second stage. The patient *screams* in an agonizing manner, and alarms those who may be with him, adding greatly to the distressing character of his sufferings. The delirium now begins to subside, or may be supplanted by *coma*. The temperature becomes lower, and the pulse loses much of its force and rapidity. The head is hot, and the respiration becomes irregular and sighing. The bowels, which were constipated in the first stage, still continue so, and the tongue is coated with a dirty-white fur. There may be *convulsions* at this time, which Ramskill<sup>1</sup> says may precipitately throw the patient into the third stage, which is one of *collapse*. This stage may resemble that of advanced typhoid. Sordes on the teeth, pinched features, dark circles about the eyes, fluttering pulse, great prostration, with loss of muscular power, dilated pupils, stertorous breathing, and the *unconscious passage* of *feces* and *urine*, are all forerunners of death. Should the force of the inflammation be exerted at the base, the symptoms are much more violent, and paralyse of cranial nerves are not uncommon.

**Causes.**—In considering the predisposing causes of acute meningitis it will be well to inquire what are the influences of sex and age. The reports of the New York Board of Health show that during the years 1867, 1868, 1870, 1871, 1872, and 1873 there were 4321 deaths from meningitis in the city of New York, 2506 of whom were males, and 1815 females; 3434 were children under 5 years; of these 1873 were males, and 1561 females. It will therefore be seen that males are more often affected than the other sex, and that the large proportion of cases occur among children.

Rilliet and Barthez take an opposite view of the matter, and consider the disease to exist more frequently after the fifth year. My own experience and the Health Board's statistics lead me to think that after this period of early life, the adult cases are comprised in the interval between the twentieth and fiftieth years, and I am unable to find the records of many cases after the sixtieth year, and am therefore disposed to believe that the disease is rare after that time. Various predisposing causes give

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<sup>1</sup> Article in Reynolds' System of Medicine, p. 369, vol. ii.

rise to the affection, and none, I think, plays a more important part in the production of the adult variety than continued dram-drinking and hard work in warm places. Over-use of the mental powers, and various disorders, such as syphilis and gout, are favorable to its development.

Croupous pneumonia, acute rheumatism, diphtheria, extension of disease from the tympanic cavity, blows upon the head, and sudden changes of temperature of any kind, are the direct causes of acute meningitis. In one of my cases the disease was the result of a sea-bath. The patient, after bathing, sat for some time with uncovered head upon the beach exposed to the heat of a noonday sun. Haeddeus<sup>1</sup> reports a case of this disease which resulted from typhoid fever.

**Diagnosis.**—Acute cerebral meningitis may be mistaken or confounded with cerebritis, typhoid fever, or delirium tremens. The delirium, headache, and disorders of motility are much less marked in cerebritis than in acute meningitis, and it must be remembered that the pulse in the latter disease is much more rapid and full, and the temperature much higher.

Typhoid fever is symptomatized by elevation of evening temperature, diarrhoea, abdominal tenderness and tympanites, muttering delirium, and the presence of petechiæ. Delirium tremens may be occasionally confounded with the disease under discussion, but it must be remembered that the history of alcoholism—peculiar delusions and alcoholic delirium, the absence of headache and the condition of the skin, are all evidences of delirium tremens, which are not to be mistaken.

**Pathology and Morbid Anatomy.**—When the pia mater and arachnoid become the seat of inflammation, we may roughly group the lesions and consequent symptoms into two classes, one indicative of basal trouble and the other of vertical. In the former, cranial nerve-trunks will be injured or diseased; while in the latter, the investing membranes of the cerebrum will be the seat of morbid action, and the functions of the cortex must be consequently destroyed, so that the symptoms will be more of a psychical character than when the base is involved.

The recent investigations and contributed cases of Landouzy,<sup>2</sup> of which 104 are presented by this author, demonstrate the connection between certain symptoms and lesions of the description to be hereafter mentioned, involving those portions of the cortex containing the centres of Hitzig<sup>3</sup> and Fritsch. These prove very clearly that violence of the inflammatory process in certain places may be attended by certain paralyses or contractions of limbs which are innervated from these centres. A case which recently came under my observation is one of this kind, and possesses great pathological interest.

E. B., aged thirty-six, born in Ireland, by occupation a blacksmith, is a stout, well-made man of nervous temperament, and up to the commence-

<sup>1</sup> Berliner Klin. Woch. 1869, p. 564.

<sup>2</sup> Contribution à l'étude des Convulsions et Paralyses liées aux Meningo-encephalitis fronto-pariétales. Paris, 1876.

<sup>3</sup> Reichert and Du Bois Reymond's Archives, 1870, Heft 3.



ment of his present trouble had enjoyed uninterrupted good health. He has not had syphilis, and his habits have been good. His mother and father are dead, the former having died of old age and the latter of phthisis. There is no family history of insanity, epilepsy, paralysis, nor of any organic nervous trouble whatever. Ten years ago, while working upon a fire-escape, he fell to the ground, two stories below, striking upon his head and shoulder. He was taken up unconscious, and remained so for fourteen hours. The only injuries he received were two severe scalp-wounds, one of which, from its slowness in healing, must have been attended by some bone injury, for he was unable to resume work until three months later. He says that purulent accumulations took place, and that "the doctor lanced them." Two cicatrices are now visible, one of which is about an inch and a half long, and is situated on the left side of the head and covers a depression about three-quarters of an inch in diameter and one-quarter of an inch in depth, the centre of which is about one and one-half inches below the median line, five inches above the left ear, and four and three-quarters inches above the centre of the left supra-orbital arch. This is the only depression visible, and the injury on the right side was apparently very superficial.

He gives no history of serious head symptoms, and when he resumed work was in good condition, there being no paralysis. About three months later he noticed a tremulousness of the fingers of the *right* hand and afterwards of the arm of the same side. There was no pain nor loss of power, but simply a marked tremor whenever he attempted to do anything. This difficulty increased to such an extent that he was obliged to resign his position as first-class workman, and become a helper, using his other arm to work the bellows. About six months after this the tremor affected the right leg, and he was obliged to leave his work.

*Present Condition*—The patient does not complain of head symptoms, except a slight hyperæsthesia of the right side of the face, of short duration. Vision normal; fundus of either eye presents no abnormal appearances; pupils respond well to light, and are of equal size. Hearing unaffected. No tremor of face or tongue, speech unembarrassed, memory good, and no intellectual trouble whatever. He has never had headache.

*Upper Extremities.*—Left side unaffected. The right hand and arm are perfectly quiet during inaction, but when the most simple voluntary act is attempted they become agitated by a fine rhythmical tremor, which becomes more marked as the accomplishment of the act requires greater nicety of coördination. When he is asked to carry a glass of water to his mouth, he spasmodically grasps the vessel and carries it upward, the elbow being raised, the tremor meanwhile increasing until the mouth is reached, when the movements become so violent that he is unable to place the rim of the glass between his lips. Certain motions are almost entirely unattended by tremor. He can extend the arm and hand, or can hold them rigidly upright, and is able to pronate the hand, but movements of flexion are attended by increased violence of the tremor. Tactile sensation is



somewhat impaired, but susceptibility to painful impressions is not diminished. There is absolutely no loss of muscular power, no atrophy of the hand or arm, the thenar eminences being covered by firm cushions, and the interosseous spaces being well filled.

*Lower Extremities.*—The left leg, like the arm, is in no way affected. The right leg, however, is agitated by muscular tremor when he attempts to use it, or approximates it with its fellow, as in standing erect. There is no loss of muscular power, but some anæsthesia, the patient being unable at any place to distinguish two points of the æsthesiometer, unless they are separated at least eight centimetres.

When he stands with his eyes closed he is “groggy,” but does not fall. He can stand upon the right foot alone, but not upon the left. When he walks, the right heel is brought down first, so that the heel of the shoe is much worn. He has some plantar formication and coldness of the foot. He has suffered from pains of a pseudo-neuralgic nature in the right shoulder and right thigh, which were centrifugal, as well as some pains which darted from the heel up the inner side of the leg. The pains in the upper extremity are not so frequent as they were a year ago. There has been no history of body-constricting band, pain in the back, or vesical trouble of any description, but for the past five years he has been constipated and obliged to take purgatives. There are no contractions whatever.

The peculiarities of this case seem to be the unilateral tremor (not disorderly movements) excited by voluntary exertion, its predominance in flexion, while certain movements of extension are almost unattended by any embarrassment, the absence of muscular weakness, contractions, or atrophy, and the evident dependence of the trouble upon a localized cerebral injury of the opposite side, which probably resulted from the fall.

I am unable to arrive at any conclusion which would lead me to consider the symptoms due to cerebro-spinal sclerosis, or one-sided posterior spinal sclerosis, if the latter anomalous condition could exist. The utter absence of loss of power and permanent contraction of the affected limbs, and the non-extension of the affection to those of the other side of the body within ten years, are sufficient to invalidate such a diagnosis.

The non-occurrence of convulsions and other symptoms of cerebral tumor renders this as a cause of the tremor quite improbable.

Of course the assumption that this patient's symptoms are due to some irritative meningeal or cortical lesion must be based upon purely theoretical grounds, but the features of the case convince me that such a condition of affairs is by no means improbable. If we refer to the charts of Hitzig and Ferrier, we shall find that they have located a cortical region which is “situated on the ascending frontal, just behind the upper end of the posterior extremity of the middle frontal convolution,” which “is the centre for the movements of the hand and forearm in which the biceps is particularly engaged, namely, supination of the hand and flexion of the

forearm."<sup>1</sup> Again, if we consult the admirable article of Turner,<sup>2</sup> we shall find very useful hints which will enable us to lay out the exterior of the cranium into regions corresponding with the convolutions beneath. One of these areas, which has been called the upper antero-parietal space, includes the ascending parietal and ascending frontal convolutions, and an injury at the point I have located in describing this case would be just over the centre, which, when experimentally irritated, produces movements of flexion and supination.

It is quite reasonable to suppose that this irritation occurring with volitional movements is due to a natural increase in the blood pressure during mental activity, a consequent increase in cerebral volume, and a resulting meningeal contact with the depressed portion of bone, which probably does not impinge upon the cranial contents at ordinary times.

Dr. James B. Ayer<sup>3</sup> reports an extremely interesting case of cerebral syphilis, the prominent feature of which was the presence of hallucinations of hearing, the lesion being syphilitic meningitis, evinced by great pain confined to the back part of the head, and psychical symptoms of interest, such as sluggishness of intellect, unreasonable dislikes, and insane hallucinations of hearing. The autopsy revealed a significant condition of affairs, namely, a patch of induration of certain occipital convolutions which bears out the statement of Ferrier that auditory disturbance ordinarily follows lesion of this part of the brain.

"Both tables of the skull were somewhat thicker than usual, at the expense of the diploe. The calvarium was heavy and dense; in other respects normal. The dura mater was ordinarily transparent. A recent coagulum was found in the longitudinal sinus. There was nothing special in the pia, except that a patch, the size of a half dollar, over the upper occipital convolutions of the right side was adherent to the brain.

"The middle cerebral artery of the right side contained a small spot of chronic endarteritis, which had diminished the calibre of the vessel about one quarter. There was a similar patch in the basilar artery, of somewhat larger size. The intima ran smoothly over these projections. On section they were found to consist of a yellowish-white, opaque tissue, and presented a marked contrast to the surrounding healthy tissue. The convolutions were somewhat flattened; the ventricles contained a trifle more fluid than normal.

"Near the longitudinal fissure, in the upper part of the right occipital region, between two occipital convolutions, there was an indurated portion of brain corresponding to the patch of meningeal inflammation. The gray matter was found atrophied to one half its normal thickness. The neuroglia in the white substance beneath was increased, and the white substance exhibited a grayish tint, but nothing else abnormal."

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<sup>1</sup> Functions of the Brain, page 307.

<sup>2</sup> Journal of Anatomy and Physiology, vols. xiii., xiv., November, 1873, May, 1874

<sup>3</sup> Boston Med. and Surg. Journal, Sept. 19, 1878, page 363.

In the majority of cases the inflammation begins at the base and extends upwards. The temporal lobe may often be its starting-point, while in other varieties the meninges covering the cerebellum may alone be involved. The appearance of the cranial contents cannot be mistaken, the membranes are red, hyperæmic and attached to each other, and the arachnoid cavity contains a considerable quantity of serum. The fluid in the ventricles is increased and may contain pus, and the choroid plexuses are found to be turgescient and enlarged. It may be stated upon the authority of Huguenin<sup>1</sup> that in some cases the ventricular fluid is purulent on one side, while it may be simply serous on the other. In aggravated cases the quantity of pus may be considerable, and if the meningitis be of the basilar form the pia mater of the base will exhibit extensive purulent infiltration. The ependyma of the ventricles may be thickened granular, and contains yellowish deposits. In cases due to traumatism, or extension of other diseases, there may be found evidences of caries or fracture. The cortex in nearly every case of meningitis of the convexity is found to have undergone decided softening, and when the meninges are removed, some of the superficial brain-substance is carried with them. Microscopic examination will reveal cortical changes of more or less recent date. The vessel coats are shrunk or hard, and areas of sclerosis, or on the other hand breaking down, are to be recognized.

**Prognosis.**—We should always hesitate in expressing our opinion as to the course of the disease, although so few cases get well that it is almost safe to say that our patient cannot recover. The prognosis of syphilitic meningitis is by no means hopeless. There may be a gradual return to health characterized by occasional exacerbations of pain, mental listlessness, etc. If the patient improves after the first week, we may consider the prognosis much more hopeful, but there are often deceitful lulls which may mislead the medical attendant. <sup>2</sup>Dr. S. G. Webber reports a case in which there was a return of intelligence just before death, which, however, was temporary. If active treatment produces beneficial results, his chances are better, while any evidence of ocular trouble, and consequently basal involvement, lessens the patient's chances materially. Should the disease result from extension or inflammation of the temporal bone, the prognosis is also grave. Death may occur in four or five days, or even in a shorter time, but the duration of the disease may extend to the tenth day.

**Treatment.**—Two indications are to be met promptly: one the abstraction of blood; the other, cold to the head. When the delirium is furious, temporal vessels swollen, and the pulse hard and bounding, abstraction of blood from the arm is to be immediately resorted to. A suggestion made by Holland many years ago is one of value, notwithstanding the fact, that it has been almost forgotten and generally disregarded. I allude to the application of leeches to the hæmorrhoidal veins; to use his

<sup>1</sup> Ziemssen's Encyclopædia, vol. xii., translation.

<sup>2</sup> Bost. Med. & Surg. Journal, Vol. ci., p. 361.



words: "I know of no mode in which a given quantity of blood can be removed in equal effect in cases where it is required."<sup>1</sup> Cold to the scalp either by ice-bags, or by a bladder filled with pounded ice, or an arrangement of rubber tubes, should be employed, and will be found to very speedily relieve the pain. Accepting a hint from Dr. Chamberlain, of this city, I have had constructed, and have successfully used an apparatus such as I will describe. It consists of a long piece of rubber tubing wound upon itself and securely held in its spiral form by tape, forming a skull cap. The upper end is connected with an ice-cooler or a cold water tap, should there be one in the apartment; and the other is fitted with a stopcock so that the discharge of water may be regulated. By this means the patient's head can be kept cool and his bed dry and comfortable, an impossible state of affairs where the douche is used. Iodide of potassium in large doses has been given with excellent effect, and its efficacy in this disease has been praised by Flint, Alonzo Clark, and others. Aconite, ergot, and the bromides are all efficient remedies in depressing the pulse and quelling the delirium; and elaterium, saline cathartics, or the old combination of salts and senna may be of service. Blisters applied behind the ears and to the neck are excellent adjuvants. Should the patient's strength be reduced, as is the case in the later stages, the free use of stimulants, nourishing food, such as milk, egg-nog, beef-broths, and nutritious but digestible food, are of great importance. In the other forms presently to be alluded to, we should be governed by the existence of rheumatism, or the advanced age of the patient, and for the former prescribe alkalies, colchicum, and other remedies of the same nature, and for the latter a generous diet and a liberal use of stimulants.

### RHEUMATIC MENINGITIS.

A form of inflammation of the meninges may be connected with, or occur during the course of acute articular rheumatism, or again it may be found without any coexisting joint trouble.

Trousseau<sup>2</sup> has described three forms of cerebral rheumatism. One of these he calls apoplectic, and it is symptomatized by coma without paralysis; a second form, first described by Gosset, is that in which delirium is followed by coma; and there is a third in which delirium makes its appearance in the course of inflammatory rheumatism. Its co-existence with joint-trouble is by no means the rule, though the majority of cases reported have been of this character. Posner<sup>3</sup> reports a case in which the inflammation left the joints and attacked the meninges. Pain in the head, delirium, and slow pulse were the prominent features of the patient's illness, and recovery took place in about two weeks. The symptoms of an ordinary attack of metastatic rheumatic meningitis are these:

<sup>1</sup> Quoted by Solly. *The Human Brain*, etc., page 353.

<sup>2</sup> Schmidt's *Jahresbericht*, vol. 113, p. 25.

<sup>3</sup> *Encephalopathia Rheumatica*, *Ibid.*, vol. 104, p. 167.



Either during an attack of acute rheumatism, or afterwards, the patient may become dull and stupid, and delirium makes its appearance. This delirium is of a violent character, and during its existence the patient may have delusions and hallucinations of sight and hearing. In a case reported by Mesnet<sup>1</sup> the delusions of persecution were a prominent feature, but there is no regularity in this mode of expression. There is usually but a slight rise of temperature, though it may sometimes attain an elevation of 106°, or thereabouts, and the pulse at the same time becomes very rapid and full. Headache of a very severe variety, such as I have described when speaking of the other forms of acute meningitis, may be present throughout the illness, and, after several days, choreiform movements may occur, and with their advent the delirium, which was before inconstant, but now becomes continuous. These choreiform movements are such as a nervous embarrassed person would make in health when suddenly disconcerted. There is an uneasy opening and closing of the fingers, and the arm is jerked backwards and forwards. The patient now finds considerable difficulty in swallowing, portions of food remaining in the mouth for some time. Great prostration and collapse may supervene, and he dies in a comatose state, or, on the other hand, there may be slow recovery, the mental symptoms being the last to subside.

Vomiting and early headache, which are so characteristic of the other forms of meningitis, are absent. Recovery is rare, and of thirty-nine cases reported by Vigla,<sup>2</sup> thirty terminated fatally. Should the patient survive, he is very apt to become insane, the variety of such mental trouble being chronic mania. Huguenin<sup>3</sup> considers that the connection of meningitis with rheumatism is threefold with respect to pathological changes:—

“*a.* Endocarditis is the connecting link, so that the combination is rheumatism, ulcerative endocarditis, meningitis.

“*b.* Purulent inflammations of the serous membranes form the connecting link, endocarditis being present or not, as may be. In this case, purulent meningitis is secondary to purulent inflammation of the serous membranes; this is very rare, and the exact connection is unknown.

“*c.* Meningitis complicates rheumatism without there being any purulent deposits in the body, or any affection of heart; the connection here is also obscure.”

Da Costa<sup>4</sup> is inclined to refer the brain symptoms in cerebral rheumatism to two agencies, the first of which is circulation of vitiated blood, and the second is the disturbance of cerebral circulation dependent upon the plugging of small arteries by fine embola, and he consequently considers cerebral rheumatism to be a disease which is not essentially an inflammation of the cerebral meninges.

<sup>1</sup> Archives Générales, June, 1856.

<sup>2</sup> Actes de la Soc. Méd. des Hôpitaux de Paris, 1865, 3me fas.

<sup>3</sup> Op. cit. p. 624.

<sup>4</sup> American Journal Med. Sciences, Jan. 1875.

A case of rheumatic meningitis which recovered under the use of cold baths—and was treated by M. Féréol,<sup>1</sup> of Paris—is the following:

The patient was thirty-four years old, of quiet and temperate habits, who was suffering from acute articular rheumatism. He was treated at first with emetics, sulphate of quinine, and colchicum, but in five days he was seized with delirium, and dyspnœa, and at the same time the pains in the joints disappeared. The temperature of the body rose to forty degrees (Centigrade), and leeches, calomel, and bromide of potassium were given without success. The temperature rose further to forty-one degrees, and blisters were placed on the scalp, and digitalis was given. There was then a little more rest, but the aspect was typhous, with stupor and continuous sub-delirium; sleeplessness, agitation of the muscles, subsultus tendinum, dry tongue, etc. After some consultation with other physicians, it was determined to try the effects of cold baths as the only remaining resource. This plan was pursued for a whole week, the patient remaining under close observation the whole of the time, and the thermometer being almost fixed under the axilla. As soon as the temperature rose to 39.5° the patient was plunged into a cold bath. From the 25th of February to the 3d of March sixteen baths were administered at a temperature varying from twenty-one to twenty-five degrees (Centigrade), and the duration of each bath was twenty minutes on the average. The patient always raised the temperature of the water from one to two degrees, and, on leaving the bath, his own temperature fell to thirty-six degrees. After several fluctuations and much anxiety on the part of the medical attendants, the patient eventually recovered completely.

### MENINGITIS OF THE AGED.

According to Prus,<sup>2</sup> meningitis of very old persons rarely presents the same symptoms as do the forms of early or middle life. In the morning the old man or woman is stupid, but conscious; speech is thick, and there is general headache and moderate fever. The warmth of the body is nearly normal, except at the head, where it is markedly increased. In the evening it is elevated.

The eyes are injected, and there is low delirium. Incoherence and restlessness, during the night, and an uneasiness which is expressed by walking about the house and going from one bed to the other, are manifestations which are characteristic.<sup>3</sup> If the disease is to end fatally, the patient becomes comatose, and dies within a week, or twenty days at the longest, from the commencement of the disease. These patients very

<sup>1</sup> Bull. Gén. de Thérap., Mar. 30, 1875. Med. News, 1875.

<sup>2</sup> Quoted by Grisolle, vol. i. p. 430.

<sup>3</sup> Ramskill speaks of the eccentric behavior of these patients, who may use the spittoon instead of the chamber pot, or commit other violations of decency. In one case which came to my knowledge, the patient urinated against the bed-post, and went about the house with his trousers always unbuttoned.

often suffer for some time before the actual attack, when there may be partial paralysis, slight wandering of the mind, and insomnia. The general indications for treatment of the other forms are applicable in these cases.

The mental disturbances are those of senile dementia, and are distinctly asthenic. The old man is querulous and irritable. He delights to talk of his early life, but cannot tell you what has occurred within a few hours. If the condition be profound, he will sit quietly by himself, groaning and complaining. He goes frequently to stool, or, more commonly, unconsciously passes his feces and urine.

### ACUTE GRANULAR (TUBERCULAR) MENINGITIS.

Dr. Robert Whytt<sup>1</sup> was the first to describe this disease, and so satisfactorily did he do so, that even after a hundred years there is very little to add to his accurate description. We shall have to study the disease as occurring in two different ways. It may be primary, and have a doubtful tubercular character, or may occur in connection with some thoracic or abdominal disease, and like the other forms of meningitis, may be confined to the base or convexity.

**Symptoms.**—Though many of the symptoms are the same, there are a few points of difference, which are the following:—

#### *Predominant Indicative Symptoms.*

##### BASAL.

Vomiting, constipation, infrequent or irregular pulse, unequal pupils, strabismus.

##### VERTICAL.

Convulsions with intervals occupied by tremor, twitching of limbs and muscles of the face, turning of thumbs in on palms, clenching of fists, frequent pulse.

When the base is involved, the symptoms may be grouped in three stages, which run their course in from four to twenty-four days. The child may be puny and delicate. He may lose flesh and complain of headache. His skin may be white and waxy, and there may be a tendency to flushed cheeks, loss of appetite, and capriciousness about food, and at night he does not sleep soundly, but starts and cries out. I have known children to seek the companionship of some other member of the family, fearing to be left alone. The child may moan in its sleep, grinding his teeth and lying with eyes widely opened. During the day he is disinclined to play, and seeks some quiet place in which to fall asleep or remain by himself. Study is irksome, and so are all other forms of mental application. Irritable or languid, he attracts the attention of the

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<sup>1</sup> Works of Dr. Whytt, Edinburgh, 1768.



mother by his behavior, which is so markedly changed. During this period I have found that headaches and crying-spells are not uncommon precursors of the actual acute disease, which may begin after two or three months.

Marshall Hall,<sup>1</sup> in his description of the hydrocephaloid diseases, alludes to the importance of vomiting as an early symptom. "The most frequent and formidable in appearance . . . is vomiting. Never, never allow vomiting in an infant to pass without paying the utmost attention, and making the strictest inquiry in reference to the functions of the brain." Vomiting is generally the first and most important symptom, and convulsions are next in importance, but these two may be associated or appear alone. Vomiting may be frequent, and is nearly always accompanied by an aggravation of the symptoms of the premonitory stage. Headache and increased temperature are present, and are very decided evidences of the gradual development of the trouble. When we arrive at this stage, which lasts two or three days, we may expect the appearance of the following symptoms: A marked rise of temperature, say from 101° to 105° F., with greatly increased pulse. The bowels are still constipated, and there is but little appetite. The patient is delirious at night, and shrieks, cries, and tosses continually. At about the sixth or seventh day of the disease, there are various local troubles, such as unequally dilated pupils, slight strabismus, but no actual loss of consciousness as yet. There is a slight increase in the evening temperature, and the pulse is irregular and ranges from 110 to 120. The tenth day finds him much worse; his excited condition being supplanted by one of stupidity. He does not recognize those in the room, and is utterly indifferent to the kind attentions of his mother or nurse. When the finger is drawn across the skin it leaves a vivid red mark, which has been considered one of the strong pathognomonic signs. The pulse is greatly accelerated, and perhaps reaches 170, while the temperature may be found to be 104° or 105°. His condition during the tenth and eleventh days is very little changed, though the apathy is if anything exaggerated. The belly is retracted, and his facies is highly characteristic, the patient having a worn and pinched look. The skin is dark and congested, and his eyes may be fixed and immobile, and there may be either strabismus or a rolling upwards of both eyeballs, so that a large part of the sclerotic is exposed. Substus tendinum and "picking at the bedclothes," with involuntary passage of feces and urine, are grave forerunners of a fatal termination. The pupils are dilated, the pulse small, thready, and quick, and respiration is very slow. The temperature is still high, though the surface may be cold and clammy, and just before death the pulse quickens and becomes almost imperceptible. Slight rigidity now becomes apparent, the patient cannot swallow, stertor follows, and then death. Marshall Hall<sup>2</sup> tersely

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<sup>1</sup> Lecture on the Nervous System and its Diseases, L. and E. Philadelphia, 1836, p. 92.

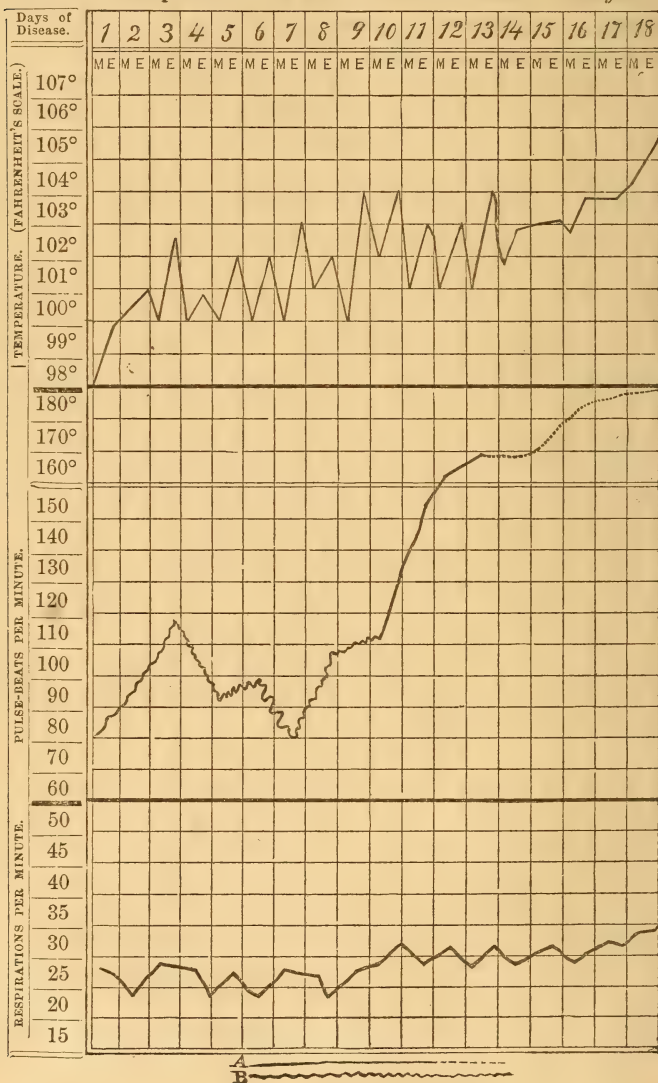
<sup>2</sup> Op. cit., p. 93.



describes this last stage as follows: "The third stage is denoted by coma and its concomitant diminution of the sentient and voluntary system, and eventually of the powers of the excito-motory system. There are

*Illustrative Chart of Temperature.*

Pulse and Respiration Variations in *Acute Granular Meningitis*.



blindness, deafness, deep stupor, absence of voluntary motion. At first the eyelids are constantly half closed, but *still* close completely on touch-

ing the eyelash. Afterwards this excito-motory phenomenon ceases. The respiration becomes irregular, alternately suspended and sighing, and at length stertorous. The sphincters lose their power, and the feces and urine are passed unconsciously." The appearance of the little patient just before death, is unmistakable. He lies with knit brow and flushed face, one side of which is drawn, while the eyes are fixed and glassy, and utterly devoid of expression.

The duration of the disease rarely exceeds twenty-four days. It will be well to dwell more fully on certain symptoms. *Temperature*.—There seems to be at first an elevation of temperature, which lasts through the first few days, say three or four, and after this time the temperature falls, until the sixteenth or eighteenth day, when it may either go much lower, or be again increased. The variations are between the normal standard  $98.2^{\circ}$ , and  $105^{\circ}$ . It however rarely reaches this high point. The surface temperature of the body is much diminished during the latter stages, but the head is always hot. *Pulse*.—Infrequent and irregular pulse is characteristic of the earlier stages of this disease, and during the last days there is increased frequency and more evenness. During the first two weeks this infrequency is to be observed, but after this it may steadily increase ten, twenty, or thirty beats more each day until at last it cannot be counted. This rule is not without its exception, and I have found intervals when both temperature and pulse would fall to the normal standard, and continue so for some days, and afterwards rise. The pulse is perhaps more rapid when the disease is being developed. I append a chart, which will enable the reader to see at a glance the condition of pulse, temperature, and respiration in a typical case. Various modifications of the cutaneous circulation have been dwelt upon by Trousseau and various writers. There seems to be an extensive disturbance of the vasomotor distribution of the skin, and when the surface is brushed or rubbed ever so lightly, or even when slight pressure has been made by the pillow, there will remain a bright red mark. This condition of the cutaneous circulation is not limited to the integument of the head, but may be present, especially towards the end of the disease, over the whole body. Trousseau<sup>1</sup> has called attention to the "tache-cerebrale," which is the name given to the appearance presented when the finger is passed over the surface, and a red line remains.

This author found that when he made cross-markings upon the abdomen, in less than half a minute the portion of skin which he had touched was suffused with a very bright red tint, which disappeared slowly, the lines made by the finger-nails remaining after the others had faded out. The regions where this redness is produced most easily are the anterior parts of the thighs, the abdomen and face. *Respiration*.—There are the usual fall and irregularity which accompany collapse of all kinds; and sighing and diminished respiration are features of the later stages. *Sensorial Disturbances*.—Headache of a deep and throbbing character is very severe

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<sup>1</sup> Lectures upon Clinical Medicine, Am. edition, vol. i. p. 877.

and continuous, lasting until coma supervenes. Various indications of the patient's sufferings are conveyed by his behavior. He presses his thumbs against his temples, or locking his fingers on top of his head, holds his head in his hands, and gives vent to suppressed groans or shrieks, holding his breath sometimes as if fearing that the very effort of expiration might increase the pain. The cry of the patient is heart-rending, but I am not disposed to agree with Trousseau that it has any decided periodicity, though there are intervals of silence. Hyperæsthesia of the scalp, photophobia, and tenderness of the muscles at different parts of the body are usual accompaniments. Bertalot<sup>1</sup> of Pfeddersheim, in an analysis of 24 cases, has found photophobia to be more commonly a symptom of the later stages, in which conclusion I am inclined to concur. The psychical symptoms are present in every case, though delirium is not so common among very young children, and when it does occur is followed by a state of semi-consciousness, and finally by coma. The patients will not speak, but rebel against food and interference of any kind, and after a time it is very difficult to arouse them. One very interesting fact is that the coma is never sudden, but is preceded in every instance by either somnolence or delirium of the muttering variety. The coma sometimes becomes less profound in character, and there may be a lucid interval before death. *Motorial Disturbances.*—The eyes are nearly always affected; and the ocular trouble is either strabismus, ptosis, or a pupillary change. The former is an early symptom, and is probably the first indication of paralysis of any kind, and is seen most perfectly when a patient is awakened or aroused. The pupils are sometimes unequally dilated, but when the coma supervenes dilatation is complete; pupillary changes are, however, by no means constant.

Unilateral paralysis is not rare; some of the facial muscles being alone affected, or there may be extensive hemiplegia, which is an advanced symptom. Spastic contractions are evidences of a condition of central irritability; and rigid flexion of the muscles of the thumb, or muscles of the sub-occipital region, are examples of this kind. The patient commonly lies with his thumbs drawn into the palm of the hand and covered by the fingers, and it is sometimes difficult to open the hands.

I have alluded to convulsions, and in addition may say, that they are more prominent in the first four days, and vary in severity if the coma be either very deep or there is a condition of semi-consciousness. In the latter case they may involve isolated groups of muscles.

*Ophthalmoscopic Signs.*—Bouchut,<sup>2</sup> Galezowski,<sup>3</sup> and numerous observers have called attention to the value of the ophthalmoscope as an instrument for diagnosis in tubercular meningitis. The latter has found

<sup>1</sup> Jahrbuch für Kinderheilkunde, B. 9, H. 3.

<sup>2</sup> Du Diagnostic des Maladies du Système nerveux par l'Ophthalmoscope. Paris, 1866.

<sup>3</sup> Arch. Gén., 1867, vol. ii. p. 262.



two forms of neuritis as evidences of this disorder; one a peri-neuritis, and the other an inflammation of the optic nerve itself. Whiteness about the papilla, deposits of granular matter in the choroid, and tortuosity of the retinal vessels, are appearances which have been described by others. Fränkel<sup>1</sup> and Steffen found tubercle in the choroid some weeks before the invasion of the disease; and Broadbent,<sup>2</sup> in examining the fundus, discovered that the optic disks were dusky red, and mottled by white spots; and the retinal veins were enlarged, while the arteries were very small. Tubercular meningitis of the convexity rarely presents ophthalmoscopic signs, though every form of convexity disease may occasionally give rise to retinal trouble.

#### ACUTE GRANULAR MENINGITIS OF THE CONVEXITY.

In the table I presented when speaking of the basal division of this disease, I mentioned the prominent symptoms of this variety. When I add that delirium and other decided psychological symptoms are highly characteristic of inflammation of the vertical region, I have described the difference between the two forms. This variety runs its course in a much shorter time, death generally resulting in from a week to ten days.

When the malady (either basal or vertical) occurs in conjunction with certain tubercular affections of the lungs or peritoneum, there are local symptoms which precede those of the meningeal disorder, but the invasion of the disease is often very sudden. Constipation, followed by a typhoid state and drowsiness, are the precursors of meningitis when antecedent lung disease has existed. Not only may children be subject to this disease, but adults are as well; and we sometimes find it as a sequence of various zymotic diseases, typhus or typhoid, remittent and other fevers, as well as pulmonary tuberculosis. A marked elevation of the evening temperature, incomplete hemiplegia, vomiting, or convulsions, are the prominent features of such a termination. Strabismus, unequal mydriasis, high pulse, and temperature, with some of the other symptoms which characterized the disease in the child, that have already been described, are generally present.

It is sometimes so insidious in its approach and development as to puzzle the observer. The phthisical patient may become listless, drowsy, or complain of headache. He often wanders and gives way to a mild form of delirium, which appears during the latter part of the day. This complication *may* occur during the early stages of the pulmonary affection.

**Causes.**—The question of diathesis naturally arises before any other, and we are immediately puzzled, for on one side we find that Rokitsky,

<sup>1</sup> Virchow's Jahresbericht, 1869, p. 621.

<sup>2</sup> Trans. of London Pathological Society, vol. xxiii. p. 216.



Robin, Empis, Clark, and others consider the disease not to be directly connected with the tuberculous diathesis, and they go so far as to question the identity of the granular deposit in the brain with tubercle; while arrayed against them are Rilliet and Barthez, Grisolle, and a host of others who are equally positive that it is in every case an expression of tuberculosis. Leaving the discussion, which is by no means settled, as the nature of the deposit needs much more investigation than it has received, we may assume that the affection is usually associated with a "scrofulous" cachexia; that it appears among children who are badly nourished, and in whom the nervous diathesis is well developed. That exposure, insufficient food, and various exciting causes, such as dentition and over-study, produce it, no one will, I think, deny. In some instances—and these are by no means few—it is impossible to find any hereditary tuberculous history. As to age, we may consider that the so-called *primary* tubercular meningitis rarely occurs after the fourteenth year, and it is probable that a great many of such cases are unattended by tubercle, but by a granular deposit of simple character; and primary tubercular meningitis in after life is, I think, a genuine tubercular disease.

Watson<sup>1</sup> makes the statement that fifty children are attacked within the first five months of life to every one after that time. I have found it to be more common after the first year, between the first dentition and the fifth year, though general practitioners who see more of these cases undoubtedly find them before that time. In large cities the mortality is undoubtedly greatest in the summer months, when diarrhoeal as well as other diseases and high temperature are conducive to its development. In the year 1871, in the city of New York, 84 deaths from "tubercular meningitis" (the reported exciting cause being "teething") are recorded in the Health Board Reports, and the greatest number were found between the sixth and fourteenth years, a fact which seems to be irreconcilable with the statement that it is generally connected with the first dentition.<sup>2</sup>

The table presented below demonstrates that males are much more frequently affected than females, and of 169 deaths 91 were of males and

<sup>1</sup> Practice of Physic, p. 270.

<sup>2</sup> An inspection of the table prepared by Dr. C. P. Russell, in the report of the Board of Health of the City of New York for 1870, will enable the reader to perceive the preponderance of mortality before the second year of life.

| Nativity. |    |        |    | Colored. |    | Under<br>1<br>Year. |    | 1  |    | 2  |    | 3  |    | 4  |    | 5  |    | 10 |    | 15 |    | 20 |    | 25 |    |
|-----------|----|--------|----|----------|----|---------------------|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| U. S.     |    | For'n. |    |          |    |                     |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |    |
| M.        | F. | M.     | F. | M.       | F. | M.                  | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. | F. | M. | F. |
| 82        | 76 | 9      | 2  | ..       | .. | 30                  | 28 | 17 | 21 | 14 | 9  | 8  | 5  | 4  | 7  | 7  | 3  | 4  | 4  | .. | .. | .. | 1  | .. | .. |

Also five males of 30, one of 50, and one of 55; this cause of death was .62 per cent. of the combined cause.

78 of females. Bertalot, already referred to, found that of his 24 cases fourteen were boys and ten were girls. Two cases occurred in the first year of life, seven in the second, five in the third, three in the fourth, three in the twelfth, and one each in the fifth, ninth, tenth, and fourteenth years. The youngest patient was ten weeks old, and twenty-two out of the twenty four were attacked between November and the end of June. The children were all more or less delicate, they had frequently grown up under bad hygienic conditions, and were generally scrofulous or scrofulorachitic. In twelve there was a distinct hereditary predisposition to tuberculosis; two cases supervened upon chronic coxitis; one upon traumatic erysipelas; two upon pertussis; one upon measles; and one upon the first signs of dentition.

There are certain physical appearances belonging to children predisposed to these forms of disease which should not be passed unnoticed. In nearly all of the cases I have seen the head of the subject was peculiarly long and large. The hair was usually silky and fine, and of light color, and in some cases hip disease and like troubles had been noted.

**Morbid Anatomy and Pathology.**—From the immense mass of confused testimony before us (for the disease has been described by nearly every writer, since the time of Hippocrates), it is extremely difficult to say whether the *post-mortem* appearances are always those of a tuberculous character, or whether the granular substance is non-tuberculous, or again whether in some cases there is tuberculous deposit and in others simple granular collections. Paisley, who, Watson says, was the first to clearly describe the affection without saying much about its tuberculous nature, has given us a very admirable collection of facts bearing upon its morbid anatomy.

Gerhard,<sup>1</sup> one of the early medical writers of this country, says: "It was not known, previously to the researches of Dr. Rufz and myself, that the tuberculous character of the disease was anything but a mere complication." Guersent, Dance, Hennis, Greene, and others shared in Gerhard's opinion, that tubercular meningitis was a "strumous" disease.

Rufz<sup>2</sup> collected 40 cases, and in every instance there was complicating pulmonary tuberculosis.

Fenwick's<sup>3</sup> tables are valuable in displaying the distribution of tubercle in the affection.

In one of these, sixteen cases of meningitis occurring in tubercular patients are detailed in which tubercle was found in the lungs and other organs, but not in the brain.

In these cases, of which ten were males and six females, there was tuberculous deposit in the lungs in every instance, and in some of them other organs were affected. Positively nothing like tubercle could be found in the brain, but this organ was either congested or anæmic. The

<sup>1</sup> Dunglison's *Prac. of Med.*, vol. ii. p. 243.

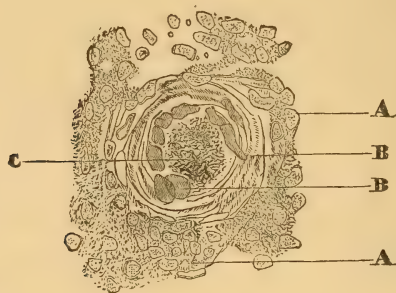
<sup>2</sup> Quoted by Marshall Hall, p. 94.

<sup>3</sup> St. George's Hosp. Reports, vol. vii. p. 35.

membranes were "wet," and the ventricles contained fluid. Four cases were under ten years of age; three between ten and twenty, and three between twenty and thirty; four were in the fourth decade, and one in the fifth and sixth. In other cases brought forward by him of general tuberculosis, it was found that of fifty-four examined, nearly four-fifths of the number were below twenty-five years. All of these fifty-four had tuberculous deposits, both in the brain and other organs.

The seat of the granular deposit seems to be chiefly the arachnoid and pia mater, though the dura mater has been found as well to be the site of granular accumulation. It is scattered mostly along the base of the brain and about the large arteries, where it may be found to consist of masses of little round pearly or yellowish bodies which may be almost as small as grains of coarse corn meal. The meningeal arteries are dotted over with these granules, and when the arachnoid is raised numerous underlying miliary granules are exposed.

Fig 12.



Tuberculous Matter about the Vessels. (Cornil and Ranvier).—A. Tuberculous deposit. B. White blood-corpuscles. C. Granular contents of vessel.

The membranes are all more or less congested and dotted with opaque spots or patches. The cortex is hyperæmic and the ventricles distended by fluid. Their ependyma is toughened and rough, and presents a granular appearance which may be likened to that of white shark's skin.

Softening of various parts of the brain, the nerve trunks and optic commissure are not uncommon evidences of the violence of the disease. Patches of false membrane which contain in their meshes these granular bodies are scattered over the convexity and base, and render the removal of the brain or its membranes separately a somewhat difficult matter. The lungs, or other organs, may also present indications of tuberculous matter.

Rendu<sup>1</sup> affirms that whenever there is paralysis of permanent form there must be some arterial obliteration from fibrinous exudation and consequent softening, and he does not believe that scattered granulations or ventricular effusion are alone sufficient for its causation.

<sup>1</sup> Review in *Gaz. des Hôpitaux*, Jan. 15, 1873.



It is rarely possible to very closely localize limited deposits before death, but occasionally this may be done.

A very interesting case is reported by Raymond which presented several suggestive points. One was that the motor centre of the right arm was the seat of granular lesion, and that there was paralysis of that member. This, then, is an exception to the rule to which I have just referred.

"The patient, a man twenty-two years of age, was admitted into the hospital in the early part of the month of January last and then presented obvious symptoms of pulmonary tuberculosis, not, however, very pronounced. The affection, indeed, seemed to be progressing slowly. He was thin, pale, coughed a good deal, and was a little feverish.

"On January 28 he began to complain of violent pain in the right hypochondrium, and two days later vomiting came on. This recurred frequently, the ejected matter having a greenish color. At the same time he suffered from severe headache, which affected chiefly the left side of the head. Fever then showed itself, the temperature rising to  $140^{\circ}$ ; the pulmonary lesions developed more rapidly, and the general condition became much worse. On March 24 he complained of great pain in his right arm, which seemed to be very heavy; at times he had great difficulty in moving it. On March 25 there were fresh pains in the arm, and motor paralysis was complete, sensibility being retained. In the evening, with a great effort, he succeeded in raising his arm to his head. The paralysis of the arm, up to the time of his death, presented the character of intermittence. There never existed any trace of paralysis in the right leg nor in the left arm or leg. Perhaps there was a slight degree of loss of power in the bucco-labial muscle of the right side, and a slight deviation of the tongue to the left, but these symptoms were a little doubtful. In the whole case, there was nothing else comparable with the paralysis of the arm, which was indisputable. The patient died on April 4.

"At the necropsy, far advanced tubercular lesions were revealed in the right lung, and the membranes of the brain were found to be the seat of tubercular granulations. These were found in the pia mater over the right lobe, and there they were disseminated along the parietal branch of the Sylvian fissure. On the left side, in addition to the tubercular granulations, there existed some meningitis with purulent deposits. The meningitis was, if it may be so said, circumscribed and localized on two convolutions, the anterior and posterior marginal near the paracentral lobe. There the tubercular granulations were very numerous, and formed a sort of tumor. The pia mater, covered with pus, adhered closely to the subjacent cerebral tissue. In other parts, where there were granulations, there was no vestige of meningitis. No other cerebral lesions, foci of softening, or obliteration of capillaries, could be discovered. There was a small amount of fluid in the ventricles, but nothing to note in the spinal cord or nerves of the arm.

"Such are the facts of this case, which may be summed up as follows: Motor paralysis of the right arm, somewhat intermittent in the sense that it was at times complete, and at other times less absolute; and to explain this paralysis no other lesion than the tubercular meningitis in the region of the motor centre of the arm."<sup>1</sup>

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<sup>1</sup> London Med. Record, July 15, 1876. Abstract from *Le Progrès Médical*, April 22, 1876.



Landouzy has collected a large number of valuable cases, showing the possibility of localization sometimes in tubercular meningitis, and has presented 43, in which partial convulsions predominated in 23 cases. In these the distribution was as follows:

The face alone, once; the face and arm, twice; the face, arm, and leg, five times; the arm alone, six times; the arm and leg, eight; the leg alone, once.

<sup>1</sup>In half of these cases the convulsions were limited, in some cases the partial convulsions were preceded by those of a general character. He was enabled to diagnose the seat of the trouble in all of these cases.

**Prognosis.**—No inflammatory disease of the brain or its membranes is more serious or rapidly fatal than is this. The termination is in death in from two to three weeks, though very rarely recovery may take place before the disease has gone beyond the period of invasion. The ophthalmoscope is of service at this time. If there be optic neuritis, and basilar meningitis is suspected, there is very little hope to be derived from such an examination; if the child recovers, it will be with impaired intellect, epilepsy, or some other serious life-long trouble.

An anonymous writer in the *Gazette Medicale* upon the treatment of tubercular meningitis, says that, in a practice of thirty years, he has seen between eighty and ninety cases, and during that time there were but two recoveries.<sup>2</sup> Bierbaum<sup>3</sup> has reported three recoveries.

**Diagnosis.**—This disease may be mistaken at different stages for several other acute conditions, viz.:—

- A. Typhoid fever—typhus fever.
- B. Scarlet fever or smallpox.
- C. Pleurisy or pneumonia.
- D. Eccentric irritation, such as that produced by worms, etc.
- E. Other forms of meningitis.
- F. Exhaustion.
- G. Syphilis.

A. Typhoid, in some of its forms, or typho-pneumonia, may resemble tubercular meningitis, either of the primary or secondary forms. This is especially the case when typhoid symptoms are added to those of phthisis. The irregular varieties of typhoid are attended by absence of diarrhoea, tympanites, and other abdominal symptoms. The eruption of typhoid may also resemble the tache cérébrale of this form of meningitis, but it is usually confined to the chest and abdomen, and is an early symptom. Typho-pneumonia may bear a close resemblance to secondary tubercular meningitis, and this is particularly the case if moist rales can be heard all over the chest, and there is some dullness at the apex; certain points are to be borne in mind, however, that will put the diagnostician on his guard. Uncomplicated typhoid is a disease of longer duration, and the abdominal

<sup>1</sup> Contribution a l'étude des Convulsions, etc., Paris, 1876.

<sup>2</sup> Gazette Médicale, 1871, 412.

<sup>3</sup> Deutsche Klinik, 1873, 184.

symptoms are usually marked. There is tenderness in the left iliac fossa, high evening temperature, nose-bleed, and usually slight head symptoms, which vary. The eruption fades away under pressure, instead of being produced by pressure or contact, as is the case in the meningeal difficulty, and the prodromal symptoms of typhoid are not nearly so marked as those of the other disease.

Typhus fever may sometimes make the diagnosis exceedingly difficult; for, as we know, its duration is about that of the tubercular trouble, and head symptoms are its marked feature. The general absence of pulmonary symptoms, the appearance of the dark rash, and the antecedents of the patient offer us guides.

*B.* Scarlet fever, which sometimes begins with vomiting and early head symptoms, may puzzle the observer. The throat trouble, the early appearance of the eruption, the peculiar "strawberry tongue" which, as far as I am aware, is found in but two diseases, diphtheria and scarlet fever, and the high and continued elevation of temperature during the eruption, are sufficient to put the medical man upon the alert.

Smallpox, without the eruption, may sometimes mislead us. The prodromal symptoms, pain in the back, vomiting, and headache, are different from the same symptoms in tubercular meningitis. They are more severe, and may immediately usher in coma. Bleeding from the nose and mouth I have witnessed in three patients. This form of smallpox is quite rare. In the course of nine years, during which I was connected with the Health Department of the City of New York, I saw over one thousand cases of the disease, and I do not remember having encountered but ten or twelve cases of this terrible form of variola. These cases were all adults. If pronounced smallpox should suggest the other affection, it will be found that in two or three days any blush eruption (which could hardly be mistaken for the maculæ of tubercular meningitis, which is a late symptom) will develop so that the characteristic vesicles may be seen. In both scarlet fever and smallpox the history of exposure often supplies the link.

*C.* Pneumonia and pleurisy can only be mistaken when we neglect to take into account the chill, pain in the side, and physical signs. The latter disease may sometimes be supposed to exist; for Gee has heard the friction sound of pleurisy in tubercular meningitis.

*D.* Reflex irritation from ascarides may produce many of the early symptoms which also indicate tubercular meningitis, and even convulsions may appear; but, unlike the tubercular disease, there is no further progress. The use of an anthelmintic will clear up the diagnosis, if we have reason to suspect these parasites.

*E.* From simple meningitis we may distinguish the disease chiefly by the late appearance of the delirium. The patient lapses into unconsciousness in the former disease in two or three days, while in tubercular meningitis the acute mental disturbance is not so immediate. Acute meningitis runs its course usually in a week.

Various intracranial diseases may resemble at different times the dis-

ease under consideration ; but as I propose to treat of these hereafter, it will be well to omit them here.

*F. Exhaustion.*—The excitement aroused in England by the Penge case gives this part of the subject decided importance. It will be remembered that one Louis Staunton, with two accomplices, one of whom was his brother, and the other a woman with whom he was living upon terms of criminal intimacy, starved to death his wife, and that they all narrowly escaped capital punishment or transportation. The coroner's jury decided that the real cause of her death was starvation, while several distinguished medical men contended that she had died from tubercular meningitis, but did not deny that she had been neglected. The disputed points seemed to be, the rapid emaciation and great anæmia of the tissues, as well as disappearance of subcutaneous fat. Her symptoms before death were drowsiness passing into coma, stertor, rigidity of one arm, and inequality of pupils. These symptoms appeared but shortly before death, and were supposed by Dr. Greenfield,<sup>1</sup> who made a most sensible and convincing communication to the *Lancet*, not to account for starvation alone, but to be probably due to tubercular meningitis.

Opposed to him are several observers (among them Virchow, who reviewed the English testimony) who held that the great emaciation, loss of weight of the internal organs, emptiness of the cavities of the heart, and certain forms of congestion were clearly indicative of starvation. Greenfield proved, I think, that none of these appearances were sufficient in themselves for us to say definitely that they were due to starvation ; that they may often be a result of exhausting disease ; that the congestion witnessed was an ordinary *post-mortem* appearance ; and finally that tubercle existed in the lungs and brain ; while in the latter there were found primary indications of softening as well as adhesion of the meninges.

Gee calls attention to forms of wasting disease with profound emaciation which may closely simulate tubercular meningitis, but are connected with digestive derangements and malnutrition ; and Sir Wm. Gull, in one of the English hospital reports, brought forward some years ago, several cases of hysterical anorexia, with emaciation ; and in the profound form of cerebral anæmia there can be symptoms which may resemble some of those expressed in tubercular meningitis so greatly, as to possibly lead to an error in diagnosis.

*G.* A case of cerebral syphilitic meningitis which closely resembled tubercular meningitis was reported by Webber. There were decided pulmonary troubles, and the *tache cerebral*, but antecedent pain for one year, mental dulness, etc., and recovery under specific treatment cleared up the case.

**Treatment.**—More can be done for the patient in the early stages than at any other time. Cod-liver-oil, phosphorus, a nitrogenous diet, and preparations of iodine are all of great service. The syrup of the iodide of

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<sup>1</sup>London Lancet, Oct. 6, 1877.



iron is an excellent remedy in the earliest stages, if we recognize the significance of the somewhat irregular group of symptoms. The iodide of potassium has been by many used during later stages. Fleming<sup>1</sup> reports a cure in the case of a girl two and a-half years old by large doses of the iodide, and the experience of others is also encouraging. Cold to the head and the bromides in the later stages are of greater benefit than any other remedies. Ergot has been successfully used by Gibney in one case of so-called tubercular meningitis. It should be administered in full doses often repeated. It will be found that a drachm may be given every three or four hours without producing any disagreeable effects, and when the disease is well developed I have been able to do more with this drug than any other, and am confident that a case of simple granular meningitis so treated by me was saved by its early and free administration. Gee recommends closure of the eyelids by adhesive plaster, should there be any ulceration of the cornea. Blistering, bleeding, and violent treatment of any kind are to be strongly condemned; quiet and darkness should be insisted upon as early as possible, and over-solicitous friends should be excluded from the sick-room. Food of a liquid form may be given by enemata, or by the mouth, using a syringe, and being careful in introducing its point between the teeth.

### CHRONIC CEREBRAL MENINGITIS.

This comparatively rare disease, which may be either the result of acute meningitis, or develop idiopathically, or after head injury, is of slow appearance and progress, and resembles several organic diseases of the brain proper, among them softening, general paralysis, and brain tumors.

**Symptoms.**—One of the early symptoms, especially of the vertical variety, is headache, which varies in severity. It is of a dull character, and is seated in the top of the head, and is increased by any effort which augments the cerebral blood pressure. In certain cases there is loss of memory, and other mental symptoms, which resemble closely those of general paralysis of the insane; and this mental impairment may terminate in dementia. Insanity is by no means a rare sequence of chronic meningitis, and may follow inconsiderable early symptoms. In an interesting paper from the pen of Mortimer Granville<sup>2</sup> seventeen cases occurred which began with sunstroke. In all of these insanity, usually dementia, followed the original trouble. The vertical form is generally complicated with encephalitis and muscular paralysis, as well as spasms and twitchings of either a limited group of muscles, or the arm and leg of one side. Tremor and sometimes convulsions occur after a short period, while after the involvement of the vertical cortical substance we may have marked motorial symptoms, such as paralysis with contractures. Paralysis of the bladder or sphincter ani, takes place, so that the patient passes his urine and feces in an involuntary manner. The disease is generally progressive, and there is an increase in the number of convulsions. The mental decay

<sup>1</sup> British Med. Journal, 1871, p. 443.

<sup>2</sup> "Brain" Part viii.



advances rapidly, and the patient finally dies, at the end of a few months, in a comatose state. The basilar form of disease is much more interesting than that of which I have just spoken, the cranial nerves being more or less involved; and symptoms of cranial paralysis of a progressive character form a distinguishing feature of the disease. Thus, in thirteen cases collected by Dr. Cross,<sup>1</sup> of this city, the third nerve was paralyzed generally on the left side in nineteen instances, and in one case the third pair on both sides was affected. In nine of these cases strabismus was noted; in five of which it was external and existed on the left side. The pupils were dilated in eight instances, and contracted once. Obscureness of vision was observed to be prominent in four cases, while ptosis existed in five, occurring once on both sides. Double vision was present in many cases. Blindness occurred once in the left eye, which was the result of suppurative choroiditis. In another instance there was loss of sight in both eyes. I may select four of Dr. Cross's cases, which represent very fully the course of the disease:—

CASE I.—A young man came to the clinic who was affected with external strabismus, ptosis, and dilatation of the pupil of the left eye. He had a most intensely agonizing pain in the head, vertigo, frequent attacks of vomiting, and paresis, if not paralysis, of the arm and leg on the same side. He was treated with mercury and large doses of the iodide of potassium. In a short time the pain in his head disappeared, and after the lapse of a few weeks the paralysis was cured. Two or three months subsequently he reappeared, with a corresponding set of symptoms in the right eye, and the right half of the body, and with pain in his head as severe as during the previous attack. He was again treated with mercury and the iodide of potassium, when his symptoms again disappeared, and have not since returned. In this case there was some slight suspicion of syphilis.

CASE II.—A man, twenty-eight years of age, came under my charge some two years ago. At that time he was suffering from pain in the head, vertigo, dilatation of the pupil, external strabismus, double vision, numbness, and slight paralysis of the opposite side of the body. As far as I was able to discern, the ocular paralysis was confined to the left internal rectus muscle. Until within a few months prior to his coming under my observation, he had apparently enjoyed excellent health, with the exception of a severe headache, from which he had suffered quite acutely. He stated that the disease with which he was afflicted had come on slowly, and gradually increased in degree. He acknowledged that he had had a hard chancre several years previously.

Under the influence of large doses of the iodide of potassium, the symptoms rapidly disappeared, and he has since had no return of the paralysis, although he afterwards experienced severe headache, which disappeared under treatment. I examined his retinae, but found no disease.

CASE III.—Shortly after this I was consulted in regard to the case of a gentleman, thirty-five years old, who was suffering apparently from symptoms similar to those observed in the preceding case, with the exception of the paresis of the extremities. He had well-marked head-symp-

<sup>1</sup> Psychological and Medico-Legal Journal, New Series, vol. ii. p. 220.

toms and numbness, which was limited to one side of the body, but the paralysis was confined exclusively to the ocular muscles. His eyes had been carefully examined by an eminent ophthalmic surgeon, who had informed him that they were healthy, and that his trouble was probably cerebral. He was a very robust man, and had apparently suffered from no severe disease until the beginning of his present trouble. On questioning him closely, he stated that he had had syphilis twelve years ago, for which he had been carefully treated, and consequently considered himself cured. When I first saw him, the double vision had existed several months, and during that time had been almost constantly present. I did not treat this patient, and consequently do not know the result.

CASE IV.—A married gentleman, forty-one years of age, came under my care in 1873. He was descended from a family saturated with rheumatism, and gout, and five of whom had died of paralysis. At this time he was suffering from myalgia, which I found to be located in the muscles of the chest and back. This condition lasted about three months, and then disappeared under treatment. He stated that prior to this time his health had been good. He had been temperate in his habits, and had never had acute articular rheumatism, gout, nor syphilis. In July, 1873, he first observed that the pupil of the right eye was much contracted. This was followed by headache, vertigo, and obscureness of vision. In December he came to my office and informed me that his ocular troubles had increased. At that time his condition was as follows: He had a dull, heavy pain behind the ears, which seemed to extend along the base of the brain, and was at times throbbing in character. There was vertigo and indistinctness of vision, which he described as a blurring of objects; his right pupil was extremely contracted, and did not respond to the stimulus of light. Far and near objects were very indistinct, and appeared to be one above the other. When he looked at the pavement it appeared to be raised above its natural position. There were double vision and strabismus.

He kept his head constantly turned to the right and downwards, in order to bring the axes of his eyes parallel. All his organs were healthy, with the exception of his brain. There was apparently partial paralysis of the right internal rectus and right inferior oblique muscles. This gentleman was, by my advice, carefully examined by two eminent ophthalmic surgeons of this city, both of whom were of the opinion that there was no disease of the eyes. An important point in this connection is the fact that this patient had been in the habit of using a magnifying glass with the affected eye to examine the delicate parts of machinery, in order to see that they were properly constructed; and this operation was conducted in a dark room, lasting several hours daily. I carefully examined this patient's spinal cord (as I always do in all these cases), but found no indications whatever of spinal disease. I ordered him to take the iodide of potassium, in fifteen-grain doses, three times a day, well diluted in water, and to rapidly increase the amount; but the first dose caused him such intense nausea and vomiting that he could not be induced to take it subsequently. He consequently ceased taking any medicine, and for some time he continued to grow worse, all his symptoms increasing in severity. He was obliged to give up his business, and has since passed most of his time in out-door exercise.

The pupil of the right eye remained permanently contracted for several months. A short time since I met him, and he told me that he was about

to resume his business, he had so nearly recovered. His pupil was still contracted, but not to the same degree that it was when he first came under my care a year ago. He now holds his head straight; there is no apparent strabismus, although his wife informs me that he occasionally sees double. His headache and vertigo have disappeared. The only medicines that he has taken during this period have been tonics and out-door exercise. I made particular inquiry in this case, in order to discover, if possible, a constitutional cause, but I was fully satisfied that none existed.

Both of these forms of meningitis may be connected with cerebral growths and syphilitic and tuberculous deposits.

**Causes.**—Males seem to be oftener affected than females, and the disease is ordinarily one of adult life. It is connected oftentimes with the tuberculous diathesis, and is not rarely dependent upon constitutional syphilis; it may be seemingly idiopathic, or result from head injury, exposure to the sun, intemperance, the acute zymotic fevers, and the other causes of meningitis.

**Morbid Anatomy and Pathology.**—The cerebral meninges have been found to be thickened, adherent to each other, or to the inner surface of the cranial bones, with effusions beneath, which have undergone partial organization; sometimes gummy exudation of syphilitic origin will be found scattered over the surface of the brain, or calcareous plates of perhaps an inch in diameter will be found in the dura mater, such as I have already spoken of in chronic pachymeningitis. If the disease has involved the cortical substance of the brain, we may discover patches of softening of variable extent and depth, and perhaps superficial abscesses. At the base of the brain the meningitis is not generally so diffuse, but occurs in circumscribed spots, the cranial nerve trunks being generally softened and bound down by bands of new tissue. In a case of meningitis following sunstroke <sup>1</sup>Granville found very interesting osseous changes.

“Calvarium strongly adherent, the plates dense; diploe obliterated; membranes very vascular, thickened and adherent to the surface of the brain along the median fissure: this was found on separation to be caused by three or four bony plates, of the size of a sixpence, with small spiculæ passing into the surface of the brain on the left side; the brain was smaller than usual and weighed only forty-four ounces; the gray matter was deficient, and the convolutions flattened and apparently not so numerous.”

In this case sunstroke was followed by headache, most intense on the left side of the head, difficulty of articulation, defective memory, and subsequent symptoms resembling those of general paresis.

**Diagnosis.**—The form of meningitis of the convexity presents so many symptoms that are common to other brain diseases, that the matter of diagnosis is often very difficult, and it is impossible at times to deter-

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<sup>1</sup> Brain, Oct. 1879, p. 314.



mine the nature of the patient's disease until after death. Meningitis of the base, however, is much more easily diagnosed. There are nearly always ophthalmoscopic appearances, which is rarely the case in the other form of disease and some one or all of the cranial nerves are paralyzed. The symptoms of tumor may counterfeit those of chronic basilar meningitis, but perhaps are more severe. If the disease be of a syphilitic character, the question of diagnosis is a puzzling one; for in some respects a condition which favors the formation of syphilitic tumor and chronic meningitis is the same, and occasionally these two diseases are found to coexist.

**Prognosis.**—Should the disease be syphilitic, the prognosis is favorable, unless the trouble be of long standing, but, if it be the result of injury, recovery is less likely to take place; should it follow the acute exanthematous fevers, there is very little hope.

**Treatment.**—Our main reliance is in the free use of large doses of iodide of potassium, or in the employment of mercurials. Active counter-irritation and the use of blisters and cauterization may afford a great deal of relief. A saturated solution of the iodide of potassium may be ordered, and the patient should be directed to begin with a dose of ten drops three times a day, and gradually increase one drop with each dose until he takes a hundred drops or more during the twenty-four hours.



## CHAPTER II.

### DISEASES OF THE CEREBRUM AND CEREBELLUM.

#### SYMPTOMATIC CEREBRAL HYPERÆMIA.

**Synonyms.**—Cerebral Congestion, temporary Cerebral Congestion (*Andral*). Hyperémie Cerebrale (*Fr.*). Hyperämie des Gehirns (*Ger.*)

**Definition.**—A condition characterized by an abnormal increase in the amount of blood contained in the cerebral vessels and expressed by symptoms which indicate pressure and irritation of the cerebral nerve cells; such increase in blood pressure being the result usually of general bodily disease.

Until a few years ago this trouble was considered as a form of organic cerebral disease, at least as a part of a morbid process terminating inevitably in softening or cerebral hemorrhage. Such is the treatment of the subject by <sup>1</sup>Andral, <sup>2</sup>Durand-Fardel, <sup>3</sup>Calmiel and many others. Notwithstanding the fact that Andral describes a "temporary cerebral hyperæmia," the condition never received any extended notice until fifteen or twenty years ago. <sup>4</sup>Schmidt describes functional hyperæmia and anæmia in his Compendium; and Jaccord, Hammond and others since have clearly established a form of cerebral hyperæmia which has not of necessity any connection with graver cerebral troubles.

Before entering into the discussion of the affection, I desire to state that in very few cases do I consider cerebral hyperæmia to be a *distinct cerebral disease*, but rather one form of expression of some general condition, and, for this reason, I prefer to use the designation symptomatic. The apoplectiform variety originally described by Andral, and many years afterwards by Trousseau, is without doubt a result of vascular rupture, and should be classed under "cerebral hemorrhage."

Symptomatic cerebral hyperæmia includes those varieties of increased cerebral blood pressure dependent usually upon diseases of the heart, liver or kidneys; such, for instance, as the symptom described by Bright as "the effect of cerebral blood pressure with venous turgescence," either functional or organic, or upon any condition which impedes the return of venous blood from the head.

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<sup>1</sup>Clinique Medicale.

<sup>2</sup>Traité des Maladies-Inflammatoires du Cerveau, tome 1, Paris, 1859.

<sup>3</sup>Traité du Ramollissement du Cerveau, Paris, 1843, p. 153.

<sup>4</sup>Compendium der Nervenkrankheiten, Leipzig, 1869.

Two forms of cerebral hyperæmia have been recognized by the majority of modern medical writers, one of them which is *active* and connected with forcible arterial fluxion, and the other *passive*, and the result of some impediment to the venous return. I prefer to adopt the terms *sthenic* and *asthenic*, as these expressions denote pathological conditions much more appropriately than do those in common use. Either may exist in a modified degree as physiological states, and it is often difficult to make the distinction between a normal process and a diseased condition; but when the cerebral fulness is constant or increased to a serious extent, we may safely judge the condition to be pathological. The division of the disease expressed by the terms I have just mentioned, though adopted by most of the authorities on nervous diseases, is for some reasons unnecessary.

Both varieties may lead to accidents symptomatized by attacks of coma, accessions of convulsion, a low grade of paralysis, mental excitement, and other serious results. These differ only in their manner of appearance. In one, they are early and sthenic expressions, and are produced by rapidly exerted and violent force; and in the other their advent is more slow, as they appear to be produced by a sluggish force or tardy impairment of cell function, though sudden accidents which embarrass the venous return may make their appearance as immediately as those of the first variety. Stupor is more decidedly characteristic of the passive or asthenic variety, than that in which rapid dynamic arterial action takes place. In this, the second variety, there seems to be a dilatation of the small vessels, a crowding out of the perivascular fluid, and consequent pressure of the distended vessels upon the hyaline membrane next to the cells, thus preventing the removal of effete material, and consequently impairing their normal action.

**Symptoms.**—The symptoms of this condition, as I have stated, may vary from evidences of what seems to be but healthy physiological function to those which are unmistakably grave pathological conditions; from simple throbbing of the temporal vessels and flushing of the face, to coma, convulsions, or mania.

Generally the symptoms are not serious, and out of the many cases I have seen (and, by the way, a large number of these mild cases are met with in private practice) they are of a type which may be recognized at once. The patient calls attention to the following troubles: A sense of head-fulness with throbbing of the temporal arteries. He may inform us that his "head seems to be of unnatural size and great weight; that he feels as if the skin covering the head is much too tight." He complains of *tinnitus aurium*, and is troubled by subjective sounds which he compares to the buzzing of bees, the ringing of bells, and the rushing of waters.

There seems to be an extraordinary acuteness of all the senses. He may inform us that there are bright specks or motes which flit across the field of vision, and may say that bright light is painful, complaining of his inability to read fine print, because the letters seem to dance upon the

page, and the words appear hazy and blurred. *Diplopia* and other visual troubles may annoy him. Sharp noises, harsh voices, and monotonous sounds seem to produce distress and discomfort. His head is hot; and Rosenthal has found that the thermometer introduced into the external auditory meatus recorded a rise in temperature. He may have hallucinations, but is generally able to appreciate their unsubstantial character. He arises in the morning unrefreshed and uncomfortable, complaining of muscular weariness, but feels better towards the middle of the day. After his dinner, particularly if it has been a hearty one, the cerebral condition is aggravated. At night he finds it impossible to sleep, and he tosses to and fro, his head being hot and his extremities cold. The mind of the patient is preternaturally active, and his brain seems filled with excited fancies and troubled thoughts—and at last he sleeps. This sleep, however, is not sound; dreams of all kinds, or nightmare, keep him in a state of wretched semi-consciousness till the morning comes to find him utterly used up. With the patient, mental exertion is irksome, and study or concentration is disagreeable or impossible. There is headache or impaired memory, thickness of speech, and various difficulties of articulation. He may substitute one word for another, even though it be one in common use and exceedingly familiar.

The emotions are generally disturbed and altered. Irritability, oversensitiveness, nervous excitement, and morbid exhilaration of spirits may make his conduct strange and unnatural to those about him; while slight things seem to disturb and harass him. The attentions of friends, though they may be of the most considerate nature, are met with explosions of temper, and the patient avoids them and prefers solitude. In such individuals in whom the condition has existed for some time, this mental change is striking. They are suspicious of their wives and best friends, and all sorts of eccentricities are indulged in. There may be a species of hysteria which prompts the individual to commit suicide, when he has no intention of doing anything of the kind. He may worry his friends by his capricious behaviour, and succeed in making every one about him miserable. Sometimes he takes violent exercise until completely exhausted, when wearied Nature asserts herself and sleep brings temporary relief.

During the progress of the disease, cutaneous numbness or twitching of some of the muscles, or even paralysis, gives the condition a serious character. The appearance of the patient is decidedly striking, and not to be mistaken. The face is red, the cheeks puffed and swollen, the eyes prominent, watery, and injected, and the conjunctivæ quite red. He is anxious and excited, or, on the other hand, stupid. The sleepy expression is one of the most valuable objective symptoms. Occasionally, in the course of the disease, there is bleeding from the nose, which may temporarily relieve the patient. The hands and feet are usually blue and cold, and so remain. After a variable period, during which the patient has presented a number of these symptoms, he may suddenly, after a hearty meal, or violent exertion or some other exciting cause, suffer an incomplete loss of



consciousness,<sup>1</sup> which is generally of short duration, and from which he can be aroused in a few minutes. When spoken to he seems bewildered and confused, and takes but little notice of what is going on about him. There seems to be incomplete loss of muscular power, more confined to one side than to the other, and he is able when less dazed to make simple voluntary movements. He seems to be annoyed by any bright light that may be let into the room. His pupils are contracted usually, and respiration is labored, while circulation is uneven, there being an irregular pulse. At first the heart's action seems to stop altogether, but subsequently it becomes quite energetic, and the pulse is bounding and full. If the attack be due to passive congestion, there may be a dilatation of the pupils, and the bloating and puffing of the face and fulness of the lips will be much more noticeable than when it is the result of the sthenic variety. During its continuance there is neither rigidity of the muscles nor stertorous breathing. The recovery is generally rapid, and after the attack there may be some epistaxis and slight mental excitement.

A form, which certain writers have called *maniacal*, may and does often occur without any of the characteristic symptoms of increased cerebral blood pressure that I have described. It is the form Milner Fothergill has so admirably described,<sup>2</sup> and characterizes usually the pathological condition, in which the nervous tissues attract an abnormal amount of blood to themselves. This variety is not necessarily connected with vascular excitement, suffusion of the face, etc. It results commonly from protracted intellectual labor and direct excitement, and the patients may be pale and bright-eyed, and active in all their movements. They are "high-strung," restless, and remarkably irritable, and at the same time are loquacious and voluble. Their thoughts and fancies seem crowded together, and are evidently originated much more rapidly than they can be expressed. "Sometimes their ideas seem to settle themselves around some prominent leading thought, the centre-piece of the rotatory chaos, while at other times there is mental excitement, with great volubility, on no subject in particular." The condition is one of exaltation, and there is a restlessness which is characteristic.

There is rarely any forcible heart action, the pulse being normal, or, if changed at all, is simply small and irritable. This condition does not seem to be confined to any particular age, though in old people cerebral congestion is disposed to take this character. The mental features may be those of ordinary acute mania, and all the phases of psychical disturbance may be expressed at some time or other. Suicidal tendencies are sometimes present. A case of this kind is reported, where the individual, during an attack of congestive mania, cut his throat. The loss of blood relieved the cerebral fulness, and his reason returned, but too late to avert the consequences of the act. This condition is one of rapid production, and under prompt treatment may disappear. Embarrassment of

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<sup>1</sup> These symptoms are, without doubt, due to small hemorrhages.

<sup>2</sup> West Riding Reports, art. Cerebral Hyperæmia, vol. v. p. 171.



speech may vary from simple awkwardness of articulation to decided aphasia.<sup>1</sup> The difficulty is rarely a serious or lasting one, and is relieved by appropriate treatment.

As I have before remarked, the second variety is more apt to be associated with deep stupor, and recovery is less certain and rapid.

There may, indeed, be a form in which profound stupor, stertor, and full hard pulse are present, and which is almost always fatal. This follows profound narcosis by alcohol or opium, and the death of the individual is preceded by involuntary discharge of feces and urine, and there is complete loss of voluntary muscular power.

Before concluding the description of the condition, it may be well to call attention to a form which is chiefly confined to early life, and occurs in the course of other diseases, or it may exist uncomplicated. In many respects it resembles meningitis. It is characterized by elevation of temperature and other febrile symptoms, among them vomiting, flushed face, headache, broken sleep, twitching of the limbs, constipation, and wandering delirium. Convulsions occasionally occur, and the attack ends in deep sleep. Recovery is the rule, although the young brain is so delicate and the violence of congestive disease so excessive, that a passive condition may take the place of, and remain after the acute condition, and death may ultimately follow. Epilepsy not rarely originates in this way. It cannot be doubted that mental worry causes cerebral congestion, and therefore accelerated action of the heart gives rise to contracted kidney and uræmic symptoms.

**Causes.**—Calmeil<sup>2</sup> and others consider that men are far more subject to cerebral hyperæmia than women, and I think clinical experience fully supports their views. Some occupations and vices of men are peculiarly apt to lead to disordered states of the circulation, while women, as it will be seen, are not affected nearly so often as the other sex, and generally suffer only at the menstrual periods or when there is a retarded flux. Andral calls attention to the symptoms complained of by women just before the time of the menstrual period—these are vertigo, flushing of the face, troubled respiration, flashes before the eyes, and other evidences which point to congestion of the brain. When the menses are irregular or suppressed these symptoms are more intense, but are promptly relieved by re-establishment of the flow. He relates the case of a man who every summer suffered from an acute train of symptoms indicative of softening, which subsided after he had had an hemorrhage from the bowels. There was no history of hemorrhoids. It is not confined to any age, but is commonly a condition of middle life, though special causes may influence its origin at other periods.

As to the etiological bearing of climate and temperature, there has been much discussion. As far back as the time of Hippocrates<sup>3</sup> we have been

<sup>1</sup> This grave form is probably due to some lesion.

<sup>2</sup> *Maladies inflammatoires du Cerveau.*

<sup>3</sup> *Aphor., Lect. iii. 16, 23.*

told that it is a condition produced or aggravated by low temperature, in which opinion he is sustained by Aretæus.<sup>1</sup> Cheyne and others consider that extreme heat favors this morbid state, and Andral contends that the greater number of cases occur in cold weather.

As far as my own experience is concerned, I have found, that either extreme heat, or cold, may develop the disease, but the greatest number of my cases have arisen from exposure to the direct rays of the sun, or have been among men whose avocation led them to pass their time in hot places. Bakers, sugar-refiners, furnace-men, glass-blowers, etc. etc., are often affected, and it is hard to say whether these people or those who overuse their brains, form the largest number. I give below a table which details the occupation of 160 of my patients.

*One Hundred and Sixty Cases of Cerebral Hyperæmia—Occupation.*

|                                    |                            |
|------------------------------------|----------------------------|
| Bartenders, or Liquor Dealers . 18 | Lawyers . . . . . 16       |
| Bakers . . . . . 15                | Musicians . . . . . 2      |
| Blacksmiths . . . . . 19           | Merchants . . . . . 15     |
| Carpenters . . . . . 3             | Painters . . . . . 2       |
| Carpet-cleaners . . . . . 1        | Physicians . . . . . 6     |
| Foundrymen . . . . . 6             | Printers . . . . . 2       |
| Harness-makers . . . . . 2         | Reporters . . . . . 4      |
| Jewellers . . . . . 2              | Tailors . . . . . 1        |
| Seamstresses . . . . . 5           | Teachers . . . . . 13      |
| Laundresses . . . . . 3            | Miscellaneous . . . . . 17 |
| Laborers . . . . . 8               |                            |
|                                    | 160                        |

By this table it will be seen that 64 were individuals whose pursuits subjected them to exposure to heat, and 54 were among persons who were hard students, worried business men, and the like.

Immediately after the heated term of 1872 I saw many patients whose cerebral condition was produced by the great heat; but the disease may be due in many instances to exposure and cold, or is at least greatly aggravated by low temperature. Perhaps a reason for this may be that in cold weather the cutaneous circulation is not so active as during the warmer season, when the sudorific apparatus requires a free capillary circulation, and for this reason there is a determination of blood to the surface. In cases of sunstroke, as we know, the skin is generally parched and dry.

As to predisposing causes we may enumerate them as follows: The organization of the individual, the existence of other disease, his habits etc. Two classes of individuals may be the subjects of cerebral hyperæmia.—those of the thick-set plethoric habit, which Reynolds calls the “lax-fibred constitution,” and those who are spare, well-knit, and of nervous temperament. These latter individuals have generally hard, rigid arteries, are past middle age, and are usually brain-workers.

In those individuals who possess a well-developed arterial system, but

<sup>1</sup> Aretæus de Signi et Caus. morbd. d. lib. 1, c. 7.

such configuration of the neck and head as to prevent venous return, there is a tendency to cerebral fulness. There are several morbid conditions which markedly influence the development of this state—malaria, renal and cardiac diseases, and syphilis being among the number. In patients with enlarged and diseased kidneys which are unable to excrete the effete nitrogenous waste from the blood, it remains in the circulation, increasing blood pressure, and necessitating excessive activity and rapidity of heart action. Hypertrophy of that organ is a result, and the walls of the right ventricle become greatly enlarged; and having much greater force than it possesses in its normal condition, it forces the blood with great energy into the cerebral vessels, and as a result there is produced the morbid condition of which we have spoken. Pulmonary disease, attended by diminished aerating space, sometimes has the same influence. Gout may be at the origin of cerebral hyperæmia; and, as I have said, malaria very often plays a very important part in the etiology.

Syphilis I have found to have much to do with cerebral hyperæmia. In this disease this condition of the cerebral vessels is not uncommon during the secondary and tertiary stages, but more often during the latter. Fournier has described a form of trouble produced by syphilis characterized by head-fulness, vertigo and attacks of unconsciousness of an apoplectiform nature, and <sup>1</sup>Chauvet thinks that such forms are but precursors of an inflammatory condition of the cerebral vessels, and that it is followed by narrowing of calibre and anæmia. Mental perturbation and hysteria seem to be connected with these forms.

An excessive indulgence in alcohol, immoderate eating and drinking or the abuse of tobacco; continued venery, and disregard of the ordinary calls of nature, are all predisposing, and some of them exciting, causes. Protracted or unnatural intellectual labor, emotional disturbance, mental strain, and intense excitement of various kinds, are additional causes of great importance.

Intellectual labor at night, particularly when there is a gas-light above the head of the patient, or prolonged business worry, not rarely favors the determination of blood to the brain. Night editors, students, and workers by artificial light are subject to this condition, and eye-strain from these occupations is a powerful factor in the causation.

Myopia and various errors of refraction and accommodation are sometimes at the origin of severe headaches of the congestive variety. Prolonged grief, especially when the patient neglects his bodily comfort, and passes long days in mourning, eating little, and gaining no sleep, is also a cause. The acute condition is not rare among nurses who have sat up at night; and they, as well as other night-workers, are very apt to combat the disposition to sleep which is healthy, by stimulants, coffee, or other agents, and after a short period a disagreeable state of congestion follows.

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<sup>1</sup> These de Concours, 1880. Influence de la syphilis sur les maladie du systeme nerveux, p. 9.



As distinct exciting causes I may mention alcoholic abuse—pressure made upon the veins of the neck by tight collars or other articles of dress—sudden exertion of any kind, such as straining at stool, or during child-birth, and lifting heavy weights. In one of my patients, the simple act of bending over to button his shoe was sufficient to produce an alarming condition of the cerebral circulation. In some persons the condition is aggravated, or attacks of the severer kind are precipitated by a visit to the theatre or some crowded place of amusement, where ventilation is bad and the room heated to a high temperature.

**Pathology.**<sup>1</sup>—Almost enough has been said to explain the changes which occur during the development of a morbid state of intra-cranial circulation. Fothergill intelligently divides the processes which may induce this condition as the following: 1. It may occur as a vascular form, with increased blood pressure, and be dependent upon extra-cranial agencies. 2. It may result from tissue alterations, in which the blood is attracted to the brain, or from toxic agents, when the two former modes are combined.

Through the cerebral ventricular connection and the spaces in the arachnoid we have reservoirs for accumulation of the fluid, when the blood pressure is diminished, and a loose and capacious receptacle in the spinal arachnoid sacs for containing this fluid when the blood pressure is above the average, so that the balance is generally preserved. When the harmony of this arrangement is disturbed, we may expect to find evidences of such inequality.

Now the question of the extent to which the brain may be compressed without injury, is one which I think will bear more discussion than it has hitherto received. Not only are the present means for experimentation

<sup>1</sup> By far the most important and interesting part of the study of brain histology is the intricate and beautiful arrangement of the perivascular space discovered by Robin\* and His,† and described by them as well as by Bastian,‡ Fothergill, and others. His demonstrates the existence of these small spaces which surrounded the vessels, than which they were several times larger. He found them in greater numbers in the gray substance, and thought he discovered a communication between the spaces in the brain and cord and certain lymph-ducts in the pia mater.

The office of these canals which loosely contain the vessels, with which they have no attachment, is a most important one; for, notwithstanding the fact that the force of blood (particularly that which goes to the cerebrum) is moderated by the tortuous course of the arteries after they enter the cranium, and their complete subdivision when they are distributed over the pia mater, the nervous substance would be little prepared without such an arrangement for sudden and violent accession of blood.

This space or cavity about *all* of the vessels enables them to expand to a great extent without any actual pressure being made upon the adjacent delicate tissues. When such a determination of blood occurs, the perivascular fluid is driven out of the nervous substance proper, and after the hyperæmia subsides, returns to the spaces about the vessels.

\* *Compte Rendu de la Soc. Biol.*, Paris 1855.

† *Zeitschrift für Wiss. Zoologie*, Band 15.

‡ Notes to translation of His's paper, *Journal of Anatomy*, vol. 1.



inadequate, but there are certain puzzling questions that come up in the most unexpected manner. The experiment of suspending the subject, constricting the vessels, and measuring the blood pressure by instruments devised for the purpose, has been tried. Dr. Loring<sup>1</sup> has related an instance where the first experiment was made, and I shall use his own words: "I would mention that a patient of mine, the acrobat known as the 'Champion Fly Walker,' informed me that in walking across the ceiling of a theatre, head downwards, he never felt the slightest disturbance in his vision, though the feat occupied fifteen or twenty minutes. This would go to show, also, that position did not have so marked an influence on the quantity of either blood or serum in the interior of the head as is now believed to be the case. For it hardly seems possible that the quantity of blood could be either increased or diminished to any considerable degree, even at the expense of the other fluids, and yet allow one to maintain for so long a time such a complete control over the faculties, especially that of co-ordination, as to perform so dangerous a feat, and one which demanded so nice an application of the senses. Be this as it may, I must say I have never been able to see the great weight of Kellie's and Burrows' experiments with animals which were killed and then suspended by the head or heels, as the case might be."

When an individual is thus suspended, we are furnished with all the external indications of cerebral hyperæmia—the flushed face, prominent eyes, etc.—but consciousness is unimpaired, and is not lost until some time has elapsed. This question is of interest, for it suggests the idea that perhaps after all many changes in cerebral function are due to the shock sustained by nerve-cells by the sudden accession of blood, and not so much to the mechanical pressure exerted. <sup>2</sup>In a very carefully prepared article by Cappie in "Brain" upon the balance of pressure within the skull, it is shown that the atmospheric pressure is exerted upon the veins as they leave the various openings in the skull, thus opposing the sudden exit of blood. He also alludes to the interlacement of vessels in the pia mater and the process of compression recurring when some of these vessels become distended. It is not difficult to realize that as a rule under ordinary circumstances the cerebral blood pressure receives no very rude modifications.

As to the value of other methods for studying the state of the cerebral circulation by gauges, watch-glasses luted into the skull, etc., I am rather sceptical. The cranial cavity is, of course, a closed cavity, and the blood supply of its contents is modified by the pressure of the bony wall. Any perforation must admit the external air, and the intra-cranial blood is then circulating under an atmospheric pressure, and I am strongly convinced such variations as have been described are not those that take in the normal state.

I have said sufficient in detailing the causes of cerebral hyperæmia to

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<sup>1</sup> Am. Psycholog. Journ., Nov. 1875.

<sup>2</sup> Brain, Part viii. 1879, p. 373.

explain any pathological processes, the description of which I may now pass over.

**Morbid Anatomy.**—Upon removing the calvarium the observer of a fatal case will probably meet with some if not all of the following appearances. Dura mater and underlying membranes injected and pink, or opalescent, and sometimes quite free from moisture, resembling in this respect a piece of damp sheepskin. The sinuses may be filled with dark blood, and the surface of the brain flattened and of a deeper color than normal. The convolutions may be flattened and pressed down so that the sulci are defined in sharp lines, the inner surface of the convolutions being pressed together. The surface of the brain, as I have said, is dark, and if the pia mater is torn off fluid blood may escape from the separated vessels. Upon making sections in a transverse plane the observer will be sometimes struck by the appearance of a pinkish blush, visible in spots, which is due to staining by hæmatoidin. This appearance, alluded to by Fox<sup>1</sup> has been compared to spots of red sand dusted on the surface. The corpora striata are of a very deep red or even violet color, and the white matter contains small puncta which are red or dark purple. The vessels are generally enlarged, tortuous, and filled with quite dark blood. Calmeil<sup>2</sup> has presented the records of autopsies in a number of cases of temporary duration. He found "in three cases that the cranial bones were notably injected; in three the vessels of the dura mater were congested; in one case there was fibrinous coagulation in the longitudinal sinus; in one the internal surface of the dura mater was furrowed by capillary arborizations; in two the cavity of the arachnoid contained liquid blood and bloody humidity; in four the cerebral pia mater was generally congested; in three cases it was reddened by extravasated blood; in one the pia mater adhered in spots to the subjacent convolutions; in one these convolutions on the right side were swollen; in four the cortical substance of the brain was generally injected and more or less colored by hæmatosin," etc., etc. We therefore must arrive at the conclusion that there is nothing remarkably significant in regard to the seat of the congestion or its form. The violence of the symptoms will, of course, be proportionate to the extent of hyperæmia, though this is not always the rule; and I have seen cases, and I think others also have, in which profound coma and speedy death were preceded by unmistakable symptoms of hyperæmia, such as contraction of the pupils, etc., and after death very slight evidences of congestion were perceptible. Microscopical examination reveals in old cases a condition which has been called by various writers "*l'Etat criblé*. This consists of a peculiar spongy, worm-eaten appearance. Arndt says that when these lymph-spaces are dilated they are filled with effete material from the brain resembling amyloid substance or leucin, called by him hyaline. The perivascular spaces are very large, and openings of some size are found at points where vessels

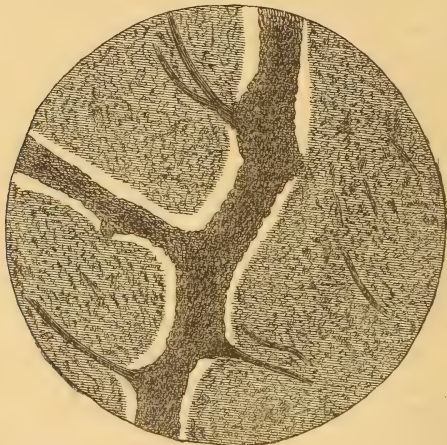
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<sup>1</sup> Pathological Anatomy of Nervous Centres, p. 55.

<sup>2</sup> Quoted by Fox, p. 56.

have been cut across. These are due to the abnormal pressure made by the distended vessel and the destruction of adjacent nervous tissue. Calmeil, Van der Kolk, Durand-Fardel, and lately Arndt,<sup>1</sup> have accounted for them as the result of œdema of the perivascular space. This appearance is a constant one in all brains where there has been continued hyperæmia, and especially in the brains of drunkards. The bloodvessels,

Fig. 13.



Distended Perivascular Spaces, with Atrophy. (Fothergill.)

when not destroyed, will be found to be tortuous and varicose, and coated oftentimes by a granular shining deposit. The pia mater is thickened, and its vessels present the appearance just described perhaps better than any other tissue.

**Diagnosis.**—The condition in its *early* stages may be mistaken for the opposite state, cerebral anæmia; in fact, the diagnosis is *always* full of difficulties.

An inspection of the following table may, however, furnish us with hints so that we may be enabled to separate cerebral congestion from cerebral anæmia. It will be observed that some of the symptoms are closely allied.

## CEREBRAL CONGESTION.

Headache (generally diffused).

Noises in the ears, generally “rumbling,” or singing.

Mental disturbance—loss of memory, hallucination.

Pupils contracted.

No heart sounds, except perhaps those of insufficiency. Pulse full.

Urine not increased, generally contains urates and phosphates.

## CEREBRAL ANÆMIA.

Headache (chiefly vertical).

Noises in the ears (generally sharp or short).

Mental disturbance—incapacity for mental work.

Pupils dilated.

Pulse irritable, aortic murmurs, sphygmographic tracing almost straight.

Urine passed in large quantities, is clear and limpid.

<sup>1</sup> Virchow's Archiv. lxiii. p. 24.



In the apoplectic, convulsive, and paralytic forms there is little danger of making a mistake.

These phenomena are sometimes liable to be mistaken for meningeal or cerebral hemorrhages, cerebral embolism or thrombosis, epilepsy, uræmic coma, etc.

The apoplectic variety may be confused with cerebral or meningeal hemorrhage. When we bear in mind that in the former there is generally almost transitory loss of consciousness and motor power, that hemiplegia is not always present, and that marked stertor is rarely found, there is no room for a mistake in diagnosis.

The other varieties of cerebral trouble, namely, embolism and thrombosis, may be disposed of by calling to mind the sudden appearance of symptoms in the former; its association with cardiac vegetations, and its permanent after-effects.

A case of this kind presents itself to my mind. A gentleman, brought to me by Dr. Asch, of New York, had been told by some friend that his nervous symptoms were due to embolism. They were these: Three months before, while sitting in his studio, he lost consciousness, and fell over upon an unfinished picture. He was conscious of his condition, but could not help himself. The room became dark, and he "saw spots before his eyes." He recovered himself in a few minutes, and resumed his work. A week ago a similar attack occurred as he was crossing the street, but he was unable to rise from the mud before assistance came. He had been worried by his business, had worked very hard, and had kept irregular hours. There was no aural disease. On neither occasion did the attack occur after a hearty meal. He had no heart symptoms at all. After each attack he recovered when he took the needed rest, and then saw no evidence of permanent trouble. The suddenness of his attack suggested embolism, but as no paralysis nor aphasia followed, and no after-symptoms remained, it seemed out of the question to consider this his disease. I made the diagnosis of local cerebral hyperæmia.

With embolism there is also generally pallor of the face, and absence of vascular excitement.

Thrombosis is a disease of slow and steady progress, with well-marked symptoms, and finally decided hemiplegia. Aphasia is also a characteristic accompaniment of thrombosis as well as embolism.

Cerebral softening can hardly be mistaken for the disease under consideration, because the former is nearly always preceded by partial cerebral anæmia, or else some distinctly inflammatory trouble. In cerebral softening there is usually local pain. Convulsive movements, paralysis, and other decided indications mark the course of the softening.

Uræmic coma may be distinguished by its deep character, and usually by an examination of the patient's urine.

The epileptic attacks of cerebral congestion resemble those of true epilepsy very closely, and in many cases we must not be too positive. There is, however, rarely any disposition to sleep, and the attacks are



generally preceded by some excitement, and are not ushered in by the cry.

**Prognosis.**—The lighter forms of this morbid condition are usually amenable to treatment, at least this has been my own experience. Of course we must be governed by the duration of the disease, the existence of other affections of an organic nature, and the age of our patient. If he be over fifty his chances of ultimate recovery are bad, but if he has not passed middle life, and the condition is directly dependent upon some exciting cause that can be easily removed, we may express ourselves more cheerfully. The existence of calcareous vessels is not an agreeable circumstance, nor the fact that he has had previous attacks of an apoplectic or paralytic nature. Perhaps the most grave prognosis is attached to the maniacal form in which the delirium is not violent nor noisy, but incessant and muttering, and in which there is a restlessness and desire for constant muscular exertion. The great danger seems to be in the continuance of the hyperæmic condition, and the possibility of its termination in cerebral hemorrhage, meningitis, cerebritis, or other organic affections. With a hypertrophied ventricle and renal disease the patient has little to expect in the way of lasting relief, and we must always give in such cases a very guarded prognosis.

**Treatment.**—Of course, the first indication, after inquiry into the patient's habits and mode of life, is to discover and remove the predisposing and exciting causes if possible. The next is to diminish blood pressure, and restore the lost equilibrium of the intracranial blood pressure both by local and general treatment.

In the majority of cases, the most simple treatment, with attention paid to the patient's bad habits, will generally remove the condition. Abstinence from alcohol in some cases, attention to the bowels, and the precaution of keeping the head cool and the neck unconfined, are the first observances to be followed by the patient.

If the condition be continued, or not relieved by these means, we may make use of several remedies, among them the bromides, ergot, and hydrobromic acid. The bromides, which were, I believe, first used for this purpose by Laycock, Clifford Albutt, and Drummond, promptly effect a diminution in arterial tension and cerebral blood pressure. Max Schuler is of the opinion that they contract the small vessels, while Nothnagle thinks their chief action is upon the nerve cells. The bromide of sodium I consider the most potent of these salts, and in doses of twenty grains, three times a day, we may expect the best results. It is well to combine it with some cardiac sedative when there is tumultuous heart action, or with some heart tonic when there is a suspicion that the heart impulse is not sufficient to properly drive the blood through the brain. Aconite in one case, or digitalis in the other, are good agents. If there be much excitement, and the mind of the individual be irritable, chloral may be advantageously administered either alone or with the bromides.

Ergot or its aqueous extract is sometimes of great benefit in these cases. Dr. Kitchen has fully described its virtues, and my own experi-

ence is directly confirmatory of what he has said. In doses of ʒj three times a day, the fluid extract may be safely administered. Squibb's or Bonjean's watery extract, in five-grain doses, may be given alone or in combination with the bromides. Should the patient be very much debilitated, for this condition is often connected with general debility, we may give strychniæ, phosphorus, iron, or quinine, though extreme care should be taken in deciding when they are useful or contraindicated.

If our patient should not be able to bear iron, we may substitute either zinc or arsenic, the oxide of the former salt being most serviceable. In the forms where this treatment is required, viz., those where there seems to be a sluggishness of the circulating blood, it is well to dispense with bromides or ergot.

During sudden attacks, local blood letting is advisable, leeches being applied to both ears, and cups over the mastoid processes. Cold to the upper part of the head, applied by means of a bladder or ice bag filled with cold water or powdered ice, is an important form of treatment. I direct my patients to apply cold to the back of the neck for fifteen minutes, every night and morning, and find that it succeeds admirably.

A drug spoken of before is hydrobromic acid, which I have found to be a valuable and powerful anæmiant.

<sup>1</sup> I first advocated the use of a solution of hydrobromic acid in cerebral hyperæmia some years ago.

Dr. Fothergill in a subsequent article confirmed my views most fully, and I have since been gratified to find how my expectations were realized by a more extended use of the remedy.

In small doses it acts very much as do the bromides, but with much more intensity. Half a drachm is fully equal to one drachm of the bromide of potassium. It differs, however, in the want of permanence of its effects, the bases of the bromic salts seeming to favor retention.

With regard to diet, and indulgence in alcohol and tobacco, tea or coffee, it is impossible to lay down any arbitrary rules. I may begin, however, by interdicting all the meats difficult of digestion, and recommending a non-nitrogenous diet. Veal, corned-beef, pork, and certain vegetables, such as cabbage, cauliflower; or nuts, spices, bananas, and other aromatic or fatty substances, are not to be thought of. Simplicity of diet is to be insisted upon. Meats should be broiled, roasted, or baked; and vegetables boiled. If the patient's comfort is dependent upon tea or coffee, it would be well to permit him to indulge in them to a reasonable extent. I do not consider tobacco the dangerous agent that it is often said to be, and if the individual be a smoker, I think his after-dinner cigar need not be cut off, and a glass or two of wine is not in the least harmful. Burgundy, Port, or other full-boiled wines should be given up as a matter of course. The abuse of alcohol and tobacco is to be looked after and stopped, if we have any reason to think that the patient has these bad habits. Open-air exercise; cold baths, with friction; or the Turkish bath, and other agents that tend to improve the cutaneous circulation, do a

<sup>1</sup> *Philadelphia Medical Times*, October 26, 1876.

great deal of good, and are to be indulged in. We must insist upon the avoidance of excitement, dissipation, and late hours and theatre-going; and it may be well to lay before our patient what may be the result of such imprudence. Should we be called in to find that the disease has manifested itself in either of the forms to which I have alluded (the apoplectic, convulsive, paralytic, or maniacal), we must order perfect quiet, darken the room, and use every means in our power to reduce the cerebral blood pressure.

### CEREBRAL HEMORRHAGE.

**Synonyms.**—Apoplexy. Hæmorrhagia cerebria (Lat.). Apoplexie cérébrale; hæmatoëncephalie; coup de sang; hæmorrhagie cérébrale (Fr.). Hirnapoplexieen, Schlagfüß (Ger.).

**Definition.**—When through disease of a cerebral vessel its walls are unable to withstand the pressure of contained blood, a hemorrhage takes place, and the nervous substance in the neighborhood is subjected to pressure, the severity of the resulting symptoms depending upon the importance of the parts which may be the seat of the accident, and upon the extent of the hemorrhage.

**Symptoms.**—I have already alluded, when speaking of cerebral congestion, to light forms of hemiplegia of temporary duration, which were dependent upon slight hemorrhages resulting from cerebral congestion. We will now deal with a form of cerebral hemorrhage of a more serious character, and it may be stated that the brain is probably more liable to hemorrhage than any other organ, with the exception, perhaps, of the spleen.<sup>1</sup>

Bastian has made the classification which I think it well to follow. He divides cerebral hemorrhage into three forms, in regard to the onset of symptoms: (1) The apoplectiform; (2) the epileptiform; (3) the simple, in which there is neither loss of consciousness, nor convulsions. The *first* may be considered as a sudden and profound loss of consciousness, which may or not disappear; but, if it does, a certain amount of hemiplegia will remain. The *epileptiform* resembles the first, but, in addition to the coma, there are convulsions. As I have said, the *simple* variety may not be connected with any loss of consciousness, the patient, perhaps, awaking in the morning and finding himself deprived of power, or noticing such a loss when some movement is attempted.

**Prodromata.**—Cerebral hemorrhage occurs generally in individuals in whom some well-developed chronic trouble has paved the way. This is the rule, although in many cases it may be the result of some recent disease. When we come to speak of pathology and morbid anatomy, these general diseases, and their influence in the production of degeneration of the cerebral arteries will be discussed; it is only necessary now to describe the forms of expression of the preparatory stages. It is not always

<sup>1</sup> Bastian: Paralysis from Brain Disease, p. 14.



necessary to look for the indications spoken of by Hughlings Jackson.<sup>1</sup> "The careful clinical observer considers minor degenerative changes, baldness, grayness of hair, the state of skin, and worn teeth. He inquires for the history of gout and intemperance."

The appearance of those individuals in whom an apoplectic effusion may be looked for, may be of two kinds. 1. The thick-necked, red-faced, and full-blooded. 2. The fair, long-necked, or aged persons, in whom the radial arteries are hard, and feel very much like strings of beads or pipe-stems beneath the skin. The existence of renal trouble also contributes to the development of an arterial state which favors rupture, and we should search for other indications of this trouble. Many of the symptoms of cerebral hyperæmia may be precursors of those that follow cerebral hemorrhage. For several days the patient may have headache, formication at the extremities as if pins and needles were being thrust into the skin, perhaps a slight anæsthesia of the arm or leg of one side; his speech may be thick and clumsy, or he may drop a word here and there, and his eyes may be red and full of tears; dizziness, muscæ volitantes dependent upon retinal ischæmia, and nose-bleed may all be indications of increased blood pressure. These last two forerunners of cerebral hemorrhage may recur at intervals for some time before the actual rupture of the vessel. The retinal trouble may be of long duration, and is of decided importance as an evidence of the degenerate condition of the cerebral vessels, and should invariably be regarded with suspicion. An atrophy of the optic papillæ with spots of blanching at the fundus, such as we find to be the result of Bright's disease, is also suggestive at times of a tendency to cerebral hemorrhage. To this list of prodromata may be added vomiting and stupor; but these are connected with so many varieties of brain disease that they may only be considered as important when occurring in conjunction with the trouble to which I have just alluded. A very serious premonitory symptom is paralysis of one limb or certain isolated muscles, which indicates organic disease. After a variable time, during which some or all of these antecedent symptoms may be observed, the vascular accident may occur. Its onset may take place in two ways: (*a*) In connection with profound loss of consciousness and suddenly. (*b*) Gradually, without loss of consciousness. We may call the first the *apoplectic attack*. Its common history is the following, and we may take as an illustrative case a male aged 50. The patient, who is of full habit, short, red-faced, and corpulent, had probably led a rather dissipated life. While reading his paper, after an unusually hearty dinner, he suddenly falls to the floor in an unconscious condition; his breathing is stertorous, the cheeks and lips being puffed out by each expiration; his face is dark, or perhaps very pale, the pupils dilated and insensible to light, and his eyeballs are fixed, turned upward, and drawn to one side. If the nostril be tickled no reflex movements follow, and the same is the case if the soles of the

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<sup>1</sup> Cerebral Hemorrhage, "Reynolds' System of Medicine."

feet be titillated. He is limp, and lies upon the floor in an inanimate heap; the pulse will be found to be hard and full, but not very rapid, and if his temperature be taken it will be probably not exceed  $97^{\circ}$ , or perhaps is half a degree lower. He is taken up and placed in bed, and after a while may make some slight voluntary movement with the limbs of one side of the body. It will be seen that the others are without power, for if the leg or arm of the paralyzed side be lifted and released it will fall to the bed as a dead weight. After an hour or two, tickling of the sole of the unaffected foot will be followed by a drawing up of the sound leg. The eyes are still rolled up and turned away from the paralyzed side of the body, and the edges of the irides are covered by the inner canthus of one palpebral commissure, and by the outer canthus of the other. The eyeballs may be sometimes slightly agitated by a feeble movement of a nystagmic character. It will be found, on removing the patient's clothing, that he has unconsciously voided his urine and feces. This condition may last for a few hours, the coma remaining profound, and the temperature rising to  $103$  to  $105$  degrees, and the pulse advancing, when death takes place; or it may be followed in an hour or two by slight signs of returning intelligence, an increase of temperature, say to  $100^{\circ}$ , with slight abatement of the regular respiration, disappearance of stertor, and the unnatural deviation of the eyes, when his temperature may return to the normal standard, and the patient so far recover consciousness as to be able to recognize those about him, and express himself by simple words, as "yes" or "no." The urine has to be drawn for a day or two, and the bed-pan used, as the bladder and rectum are implicated.

This form of cerebral hemorrhage may be connected with an epileptiform attack in the beginning, and the convulsion may be either confined to one side or be general. It would be well, before going further, to dwell upon certain elements of the apoplectic attack and analyze the symptoms.

#### THE PSYCHICAL DISTURBANCES.

Sudden compression of the cerebral mass is always attended by unconsciousness, but it is a serious fact that slowly developed growths, such as large tumors or abscesses, seem to accommodate themselves to the surrounding tissues, so that sometimes no loss of consciousness occurs whatever. I have seen a large abscess occupying an extensive tract of one hemisphere without producing the least loss of consciousness. The large effusions which produce unconsciousness are, in the opinion of Mr. Hutchinson,<sup>1</sup> productive of the psychical condition, by inducing anemia of other parts through sudden pressure. Small clots are undoubtedly productive of suspended consciousness, by cutting off either a large vessel, or by injury to some important sensory ganglion.

Consciousness is either restored through the re-establishment of the

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<sup>1</sup> London Hospital Reports, vol. iv., 1867.

blood supply or the subsidence of shock, except where the hemorrhage has taken place in the medulla. The variation in the loss of consciousness is of great importance to the physician, especially in regard to prognosis. In severe cases there may be slight improvement in this respect. The patient's intelligence returns to such a degree as to inspire his friends with some degree of hope; but there is often a sudden relapse to the original state of coma, dependent upon fresh hemorrhage.

#### RESPIRATORY DISTURBANCES.

Stertor is an important symptom, and should always be looked upon with alarm. It is indicative generally of some lesion of the base, and nearly always lasts until death, if there be a very large effusion, but disappears after a few hours if recovery is to take place. Respiration undergoes very decided modification. Hughlings Jackson,<sup>1</sup> in speaking of disturbed respiration, says: "Again, not only is the rate of respiration to be considered, but the character of the respiratory movements are to be noted. As they quicken in *rate*, so do they become more extensive in *range* though such respiration is still short. Thus in the first stage there may be only quiet action of the diaphragm, but at length the sides of the chest evert strongly in inspiration, the abdominal movement being less obvious, and at length the upper thorax takes part in the process. In severe cases the epigastrium sinks in during inspiration. This is probably partly owing to elevation of the attachments of the diaphragm from increased action of the sides of the thorax, and partly to pushing down of the diaphragm by increasing bulk of the lungs from congestion or œdema"

#### CONDITION OF THE EYES.

Prevost,<sup>2</sup> Vulpian, Lockhart Clark, and others were among the first to call attention to a peculiar diagnostic point which, though not always present, is of great value when it occurs. This has been known as "*conjugate deviation*." During the apoplectic condition the eyes of the individual will be fixed, so that they look upwards and outwards, towards the side of the lesion, and away from the paralyzed side of the body; the only exception being when the lesion is in or behind the pons. It is more often seen when the attacks are sudden, and it is a phenomenon of short duration, lasting at the most but a few days. During sleep the condition subsides, and the eyeballs are restored to their normal state, but immediately on awaking they return to this position, and in spite of the patient's effort the axis of vision cannot be changed. When the effusion is a large one, or when the onset is epileptiform, the pupils are at first very wildly dilated; but when there exists a lesion in the pons the pupil which corresponds to the sides of the lesion is greatly contracted. Unequal dilatation, however, is not of very great diagnostic value. If a lesion in the pons be extensive, both are contracted.

<sup>1</sup> Op. cit., p. 548.

<sup>2</sup> Gazette Hebdom., Oct. 13, 1865.



## TEMPERATURE AND PULSE.

Thanks to Bourneville,<sup>1</sup> we are enabled to study systematically the variations of temperature. He divides the cases into four groups: 1. Copious cerebral hemorrhage, rapidly fatal, and attended by lowered temperature. 2. Cerebral hemorrhage, terminating fatally in from one to two days, in which the temperature is primarily lowered and afterward heightened. 3. Fatal cases in which death takes place in from two to six days. In these, as in other forms, there is at first depressed temperature, next a return to the normal standard, with slight variations, and finally a decided rise. 4. Favorable cases, in which there are the primary lowering, a secondary rise, and final return to the standard of health.

These variations in temperature range between 96 and 108 degrees (rectal temperature). The pulse variation bears but slight relation to the fluctuation of the body heat. In the four classes spoken of, we may consider in the first, that the pulse is full and slow, ranging from 55 to 65. With the rise of temperature which characterizes the others, it becomes greatly accelerated, beating oftentimes 120 to 130 per minute, losing its full character, and becoming small and irritable, and if death occurs, grows gradually weaker. If recovery follows the attack, there is a gradual return to its normal rate. Of course, this must be a very unsatisfactory consideration of the state of the pulse, for the apoplectic condition is not always the same, collapse and reaction varying greatly in regard to their occurrence and duration; so the pulse, as well as respiration and temperature, undergoes many irregular modifications.

## ATTACKS WITHOUT LOSS OF CONSCIOUSNESS.

The other form, in which the individual preserves his consciousness, is not so serious a condition as that just described. The person may present some of the premonitory symptoms already mentioned, or, on the other hand, may receive no warning, but while engaged in any ordinary occupation may suddenly find one-half of his body to be paralyzed, and be unable to communicate with those about him, there being slight aphasia. With the paralysis there may be anæsthesia. This state of affairs may begin during the night, and on awakening in the morning he may find it impossible to leave his bed. The paralysis is sometimes gradual, the loss of power affecting one member, and afterwards the other, an unexpected feebleness being suddenly noticed as he is about to perform some act. One of my patients, an acrobat of dissolute habits, while preparing for the performance, found, when he attempted to put on his tights, that his right leg was quite powerless; he made an effort to stand, but became dizzy, and grasped for support a pole that was near. After repeated efforts to dress he abandoned the attempt, summoned assistance, and was taken home; the same night the right upper extremity was affected. He had

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<sup>1</sup> Etudes cliniques et thermometriques sur les Maladies du Système nerveux. Paris, 1872.

never had any previous warning. Attacks of this kind may be the forerunners of others of a more serious nature. In illustration, may be mentioned the case of S. C., a married woman, aged 41. She was drawing water at a sink, when she became suddenly giddy, and had to take hold of the banisters to steady herself. She stood thus until some friends put her into a chair and carried her to her room. She sat there that day, and was helped to bed, but did not discover her paralysis until next morning. Was not unconscious at any time of the attack. Her paralysis, when she discovered it, was somewhat worse than it is at present, and she could not speak as well as she now does. A few days after the attack she went to a hospital, where she remained one month. She entered the Epileptic Hospital July 6, 1875, and was put upon strychnine and belladonna, there existing an inability to retain her urine. I take the notes of her subsequent history from the case-book of the hospital.

"Sept. 22. At 7.30 last night it was noticed that she could not speak as well as formerly. It was quite difficult for her to speak so as to be understood. She laughed a little immoderately at her inability to clearly enunciate the words.

"An hour afterwards, in attempting to leave her bed, she fell, and since then has been scarcely able to speak, and can only say a few words. No other symptoms were noticed. Her strength of muscle and sensibility seems unaffected. She cries now continually, and seems to be depressed because she cannot speak.

"Oct. 13. Patient can tell her name, and can name every article shown her. A little thickness in articulation.

"Pupils react well. Lenses of eyes are a little opaque—the left a little more than the right. Face palsy almost passed away. Lower facial muscles act well. Sensibility in face fair. Tongue points slightly to the right.

"Voluntary motion abolished in right upper extremity, the least motion in shoulder excepted. Articulations are all flexed in the right upper extremity, and the contracture is greatest in the hand, the fingers almost touching the palm. Elbow and shoulder are less rigid.

"Extension is not painful, and there are no spontaneous pains in arm. Sensibility to contact in hand good. On finger tips feels the points of æsthesiometer at three millimetres. There is no numbness in hands. Patient considers the paralyzed hand the warmer of the two. Between index finger and middle finger of right hand in three minutes' time the temperature is  $98^{\circ}$ . Same place on left hand in three minutes' time temperature is  $98\frac{1}{2}^{\circ}$ . Right lower extremity, no motion in toes and ankles, considerable motion in knee and hips, no numbness, no contraction.

#### THE RESIDUAL PARALYSIS.

A paralysis, remaining after the "apoplectic stroke," is generally unilateral, though in rare cases, where the pons is affected at the central portion, the paralysis may exist on both sides of the body; this one-sided paralysis is known as *Hemiplegia*, and may be complete or incomplete as

regards sensation and motion. When we examine our patient after the immediate grave symptoms have to some degree subsided, we will find the limbs of one side limp, powerless, and generally without sensation; the face paralyzed on the same side, and its other half drawn up by the healthy muscles, as their antagonists are unable to perform their functions. If the patient be sensible enough to put out his tongue, it will point to the paralyzed side, while the eyes, if conjugate deviation exists, will turn in an opposite direction in a manner already described.

Jastrowitz<sup>1</sup> has called attention to a peculiar symptom, the tendency of the patient to slip out of bed on the unaffected side. This is caused by the inability of the paralyzed limb to support the weight of the sound part of the body. He also alludes to the fact, when pressure is made on the saphena nerve, at the point where the vastus externus makes a groove with the vastus internus, that the cremaster muscle on the paralyzed side will not draw up the testicle, which is not the case on the other side of the body. In other forms of paralysis, to be hereafter described, there is not the same uniformity of symptoms, there being perhaps paralysis of special cranial nerves, or those of the muscles of the face on the side opposite to the body paralysis. This variety has been called *cross paralysis*. Both sides of the face or both sides of the body may be involved, in which event there is a speedy fatal termination. Occasionally the muscles of the pharynx may be paralyzed, and sometimes the larynx. A case of this latter kind is reported by Luys.<sup>2</sup> He mentions the case of "a woman who had a sudden attack of apoplexy with hemiplegia of the left side, but with no disturbance of sensibility or of the organs of special sense. The congestive phenomena of the onset being calmed little by little, the patient regained consciousness, and stated that four years previously she had been struck for the first time with left hemiplegia, and since then had been aphonic. Her intelligence was good, and she spoke distinctly, but in a low voice. She had no paralysis of the tongue, the soft palate, or the lips. A few days later, she was seized with new congestive symptoms, and died insensible."

This laryngeal paralysis is undoubtedly a much more common affection than it is generally supposed to be, and the probability is that many of the cases reported as aphasic are in all probability aphonic. Our patient, after his return to consciousness, will then be found to be hemiplegic, and, if he is amused and attempts to laugh, we will plainly notice facial distortion, which follows any such efforts. The surface temperature of the paralyzed parts is usually higher than on the other side, and the limbs may seem to be of greater contour, and true arthropathies may be presented. This appearance has been noticed by Hitzig,<sup>3</sup> who, in referring to Charcot's cases, presents seven of his own, in all of which there was incomplete dislocation of the head of the humerus, with irregular

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<sup>1</sup> Berliner Klin. Woch., Aug. 2, 1875.

<sup>2</sup> La France Médicale, Sept. 28, 1875.

<sup>3</sup> Virchow's Archiv., xlviii., p. 345.



pains of the arm, increased by pressure. The paralyzed arm was swollen, warmer and more moist than its fellow, and the pains alluded to began about six weeks after the apoplectic attack. Hitzig is of the opinion that this condition of affairs is not directly dependent upon the central lesion. Voluntary power is lost in proportion to the extent and situation of the lesion. Should it be in the cortex or corpus striatum, a very small lesion may produce very decided impairment of motility, while such is not the case in the white matter of the hemispheres. It will generally be found necessary to draw the patient's urine for a few days, for the bladder loses its expulsive force, and, if this procedure be not resorted to, there may be retention. Electric contractility seems to be exaggerated at first in the paralyzed limbs, and a very weak electric current may provoke the most energetic contractions. In certain cases there may be an increase of reflex excitability and tactile sensibility. Sensations may be even sometimes reversed, warmth being felt as cold, or *vice versa*, or, as in the case quoted by Bastian,<sup>1</sup> a warm object may be appreciated as a weight. "A hot body on the face was recognized as pressure only; on the arm it was felt as such, though the sensation was not distinctly localized, whilst on the left leg the same hot body was recognized correctly as regards situation, though it gave rise only to a feeling of tingling." I have often witnessed hyperæsthesia of the paralyzed limbs, which were very tender to the touch. Anæsthesia generally exists, however, and electric sensibility is greatly diminished. At the end of a few days it is not uncommon to find marked rigidity of the paralyzed limbs, increased reflex excitability, and other evidences of slight cerebritis at the seat of the clot. The tendon reflex is markedly increased in the paralyzed limb, and the slightest tap will evolve an energetic contraction. Gradually there is a return to the normal condition, and articulation, which was imperfect in the beginning, may become more distinct, or, should there be aphasia, the patient will begin to command a greater number of expressions. A week or so passes, and he is able to protrude his tongue in a much straighter line than before, while the paralyzed muscles of the face slowly recover their lost power; but when the levator palpebralis is paralyzed and ptosis results, restoration is much more slow. In regard to this paralysis, Bastian has reminded us that very often deformities exist, such as the absence of teeth on one side, which may produce an appearance of facial paralysis, when in reality none exists. This is seemingly a trivial matter, but its neglect is likely to lead to grave errors in diagnosis and prognosis. As months go by, gradual amelioration of the patient's condition takes place, the limbs regain their power, the leg first, and finally the arm, and the patient may be at first able to move his toes, then to raise his leg, and, when he leaves his bed, gradually begins to acquire power of locomotion. The walk of the hemiplegic is not to be mistaken; his gait is shuffling, the toe of the boot is dragged over the ground, and the leg thrown outwards and forwards, the knee being stiff, and the

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<sup>1</sup> Op. cit., p. 128.

arm swung helplessly by the side. As the gait improves, and the patient gains more control over his limbs, he is able to perform movements which require the action of the muscles of the hip-joint, knee-joint, and finally the ankle and toes. Should he only partially recover, numerous secondary conditions may follow, as results of non-improvement of the cerebral condition. These are chiefly of a motorial character, and consist of spasms, permanent contractures, bed sores, atrophy, and inflammations of nerve-trunks. Such sequela may be called—

#### THE POST-PARALYTIC STATES.

I may enumerate these as—1. Permanent contractures; 2. Trophic alterations; 3. Tremor (post-paralytic chorea of Mitchell and Charcot); and, 4. Slow clonic spasms (so-called athetosis).

Of 32 cases of old hemiplegia seen by Bouchard<sup>1</sup> at La Salpêtrière, in 31 there were paralytic contractures. The other case presented what he called *l'hémiplégie flasque*. This form is of slow appearance, and affected in the beginning the muscles of the forearm. The fingers were flexed, and the forearm was pronated and flexed on the arm, and at the same time the humerus was drawn to the trunk.

According to Strauss,<sup>2</sup> this form presents several variations, and sometimes the hand is brought in contact with the trunk, either on its palmar, dorsal, or radial aspects. Of a large number of cases that have come under my observation, I have found that deformities of the upper extremities are much more common than of the lower; the fingers are commonly flexed and rarely extended, while the muscles of the trunk seem to be exempt from this change; and, indeed, I cannot call to mind a single instance of this kind. Contractures of the muscles of the lower extremities are apt to produce deformities which resemble talipes, equinus varus or valgus, and the toes are flexed upon the sole. Contractures of the facial muscles are quite rare, and of late appearance. The deformities are always quite striking, because of the antagonistic action of unaffected muscles, and usually no amount of force can overcome them. Trophic changes are by no means rare, either in connection with contracted muscles or alone. I have now several patients under observation who are hemiplegic. In one of these the skin of the paralyzed hand is white and puffed up; the heads of the phalanges and metacarpal bones are reduced in size, so that there is no enlargement at their points of articulation, and a consequent depression exists. In other cases there is considerable muscular atrophy to be witnessed in the palm of the hand; and in others the bones of the arm are greatly diminished in size, and the interossei quite wasted away.

Charcot<sup>3</sup> has written extensively about a form of neuritis following cerebral lesions, which is supposed to be of a central nature. That *ascending* (from the periphery to the centre) neuritis sometimes takes

<sup>1</sup> Des Contractures, Paris, 1875, p. 16.

<sup>2</sup> Op. cit.

<sup>3</sup> Leçons sur les Maladies, etc. Fasc. 1, and previous articles.

place after cerebral hemorrhage there can be no manner of doubt; and in one case, at present under observation, the neuritis began at several different peripheral points of the nerve, and there were consequent atrophic muscular changes.<sup>1</sup> The form of neuritis, however, most deserving attention is that known as *secondary degeneration*, described quite fully since the first edition of this book, especially by Flechsig, Charcot, and Brissaud. It is pathologically the invasion of the motor tracts, which extend downwards involving the pyramidal parts of the lateral columns of the cord, and, as a result, we find beside loss of motor power, the appearance of contractures and an exaggeration of the tendinous reflex. The disorders of motility are numerous, and depend more or less upon the lost or impaired inhibitory power of the individual, and the paralyzed muscles which are their seat.

Dr. Gowers<sup>2</sup> presents the following excellent table, which embraces all the disturbances of motility which may occur after the hemiplegic attack:

#### POST-HEMIPLEGIC DISORDERS OF MOVEMENT.

|   |   |  |   |   |   |   |
|---|---|--|---|---|---|---|
| Quick, clonic spasm, of<br>intermitting type. | { | Regular (continuous, or on movement)                   | { | Tremor  | { | Fine.   |
|   |   |  |   | Certain, regular, movements, due to interossei, pronators, etc. |   | Coarse.   |
|   | { | Regular (continuous, or on movement)                   | { | Choreoid  | { | Continuous spasm, or inco-ordination of movement. |
|   |   |  |   | Jerking   |   |   |
| Slow, mobile spasm, of<br>remitting type      | { | Continuous—"Athetosis"                                 | { | On movement=slow, cramp-like, inco-ordination                   | { | "Spastic contracture" of hemiplegic children.     |
|   |   |  |   |   |   |   |
| Tonic spasm, varying                          | { | Of interossei, conspicuous                             | { |   |   |   |
| Fixed rigidity, unvarying                     | { | Of flexor-longus digitorum, conspicuous=late rigidity. |   |   |   |   |

The individual retains but little of his control over the affected muscles, though voluntary power exists usually to a variable extent. The influence of the will though often increases spasmodic movements. Spasms and tremor affect first the smaller muscles, while tonic spasms affect the

<sup>1</sup> These trophic muscular and cutaneous changes are of a most interesting nature. Duncan\* found in one case that an eruption had appeared on the thigh of the paralyzed side which disappeared with the return of power; and Charcot† and Payne‡ another. In a case mentioned by the former, a vesicular eruption appeared, which followed the distribution of the superficial ramifications of the peroneal nerve, and was coincident with the hemiplegia. In this case the hemiplegia followed embolism, and a branch of a spinal artery (rami medullæ spinales, of Rudinger) was found obstructed by a plug. Pressure had been made on the spinal ganglion from which one of the branches of the sciatic originates.

<sup>2</sup> Med. Chir. Trans., vol. lix.

\* Journ. of Cutaneous Med., Oct. 1868, p. 69; quoted by Charcot.

† Op. cit., p. 72.

‡ Br. Med. Journ., Aug. 1871.



larger muscles of the limbs. One form of tremor of a post-hemiplegic character has been called by Mitchell "post-paralytic chorea;" the tremor is suggestive of sclerosis, and may begin within a period ranging from one to several months, affecting generally the upper extremities, and it is aggravated by any exercise of volition. It may affect both extremities, but very rarely the face, and the movements are quite coarse, and may be associated with a certain amount of hemi-anæsthesia. A variety of movement of a clearly post-hemiplegic character has been elevated to a distinct position, and given the name "athetosis" by Hammond. As this condition is ordinarily a secondary affection to other neuroses as well as hemiplegia, the undue prominence which it has received is entirely undeserved. Gowers says: "Neither clinical history nor supposed pathology of athetosis affords ground for separating it from other forms of disordered movement commonly seen after hemiplegia, but any one of which might occur in the primary affection." Charcot<sup>1</sup> refuses to acknowledge its distinct character. He presents several cases, all of which followed some form of hemiplegia; and the literature of neurology is replete with examples of so-called athetosis which are generally connected with hemiplegia, chorea, or even hysteria.

<sup>2</sup> Brissaud has studied the particular features of the rigidity of late hemiplegia, or, as he calls them, the "permanent contractures of hemiplegia," which are found to involve the flexor muscles. There are often what are called *associated movements*; for instance, when one of his patients was told to firmly close her left hand forcibly it was found that the movement of flexion of this hand was always accompanied by slow movement of flexion of the *right*, moreover that when she opened and shut her left hand a number of times, the right became closed in the position of true contracture. This genesis of movements in the sound side is a feature of old contracture.

The easy production of an increased tendinous reflex is always possible, and whether the tendons are lightly tapped or the member flexed or jarred there is a tetanoid state, or a series of spasms produced and the increased knee reflex commences, according to Brissaud, as soon as the appearance of secondary contracture begins. The myograph has been used to test the tendon reflex in hemiplegia. By the attachment of an ingenious instrument, constructed by Dr. F. Franck, it was possible to make some very valuable records, showing the duration of the reflex, the amplitude of the contraction and its character. <sup>3</sup>Tochirjew and <sup>4</sup>Burckhardt established the duration of the normal reflex at from 32 to 34-thousandths of a second, while Gowers believes the time to be longer. Brissaud has fixed the time at 50-thousandths of a second, as that in which the reflex occurs in the normal state.

<sup>1</sup> Op. cit., 4th part, p. 493.

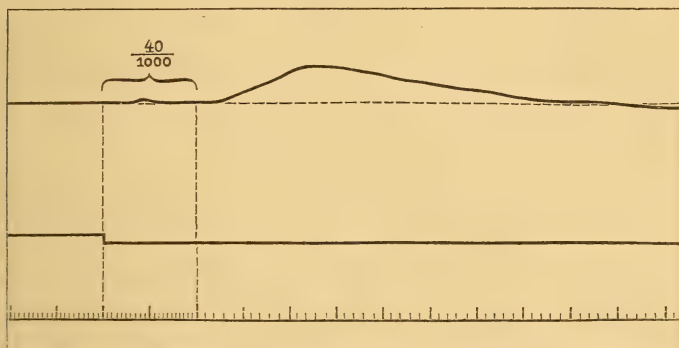
<sup>2</sup> Recherches, etc., sur la Contracture permanente des hemiplegiques, E. Brissaud, Paris, 1880.

<sup>3</sup> Archiv. fur Psychiatrie viii. Band 3 Heft.

<sup>4</sup> Centralblatt fur Med. Wissen, 1878, quoted by Brissaud.

It would be going into the subject to the extent of neglecting those of greater importance were I to do else than present the conclusions drawn by modern observers. One of Brissaud's plates shows the contraction on the healthy and contracted sides. The upper irregular line gives the contraction, the lower line the time tracings, and the time of excitation.

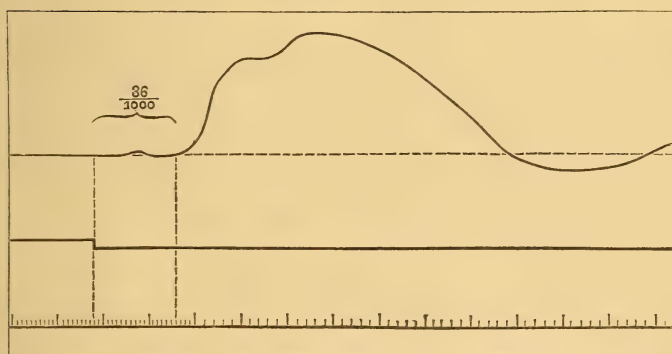
Fig. 14.



Hemiplegia with contracture. Reflex on sound side. Time of reflex 40-thousandths.

#### TRACINGS OF PATELLAR TENDON-REFLEX.

Fig. 15.



Hemiplegia with contracture. Affected side. Time of reflex 36-thousandths.

**Causes.**—Any agency which favors a degeneration of cerebral vessels leads to the occurrence of hemorrhage such as I have just described. The list of such causes is therefore a long one. Among the many formidable diseases, leading to that which forms the present subject are those of the heart and kidneys. Hypertrophy of the left ventricle, Bright's disease, and local disease of the arteries with deposits of atheromatous matter, or obliteration of vessels by softening, pressure made by tumors, and through other diseases of the brain, may be mentioned as influencing the

causation of cerebral hemorrhage. Cerebral hemorrhage is an affection of advanced life, though cases are on record among children. A careful inspection of the records of a great many cases discloses the fact that the majority are between fifty and sixty. With the advance of life and corresponding impairment of vitality, the arteries become rigid, the heart hypertrophied, and the general vascular system undergoes important changes. I have already alluded to the annular and hard character of the arteries; the *arcus senilis*, which consists of a small whitish circle which may be seen at the edge of the cornea, may be mentioned in addition as a suggestive sign, and attention may be called to the degeneration of the choroid. The color of the face is dusky red, and many of the capillaries of the skin covering the cheeks and nose are quite tortuous and dilated, and present minute varicose enlargements. As to inheritance of an apoplectic tendency, I fully agree with Hughlings Jackson, that the only heritage transmitted from father to son is the liability to arterial degeneration, gout, etc. This exception to the general rule is somewhat conspicuous, for the heredity of many convulsive and neuralgic, as well as the trophic diseases, is a well-established fact, and has long been recognized as an important etiological factor. Cerebral hemorrhage, as I have stated, is by no means confined exclusively to adult life. Numerous observers have called attention to cases which have occurred among very young children, though, in these instances, injury has generally produced the accident, especially such mechanical causes as convulsions, anæmia, etc. And now regarding the predisposing states which favor the rupture of a vessel. An hypertrophied heart, enlarged by overwork in forcing the overloaded blood which must be formed when the kidneys do not properly act as eliminants, is the first factor of the disease. With this condition of affairs the small vessels must necessarily be subjected to abnormal strain, and consequently undergo such changes as thickening or aneurismal dilatation, or even actual destruction. The arterial changes, of which I will more fully speak when we come to consider the pathology of the disease, are fatty degeneration, aneurismal dilatation, and calcification. These conditions are produced by alcohol, and improper diet, such as continued indulgence in fatty food. A sedentary life, connected with great and protracted intellectual strain, as well as such diseases as rheumatism, syphilis, and other chronic maladies, enter the field as predisposing causes. Season appears to have some influence in the production of cerebral hemorrhage, the majority of cases occurring in winter. As to exciting causes, their name is Legion. Straining at stool, coition, violent muscular effort of any kind, the indulgence in stimulants, and in fact any agency which either promotes an abnormal blood supply to the brain, or prevents its return, will have the effect, should there be disease of the vessels, of producing rupture. I have taken from my case-book data showing the causes in a number of cases, which in some cases preceded the actual hemorrhage by some hours:

|  |    |
|--|----|
| Lifting a heavy weight, or other muscular effort . . . . . | 12 |
| Excitement (alarm of fire) . . . . .                       | 1  |



|   |    |
|---|----|
| Violent exercise in drawing water . . . . . | 1  |
| Falls . . . . .                             | 4  |
| Fright . . . . .                            | 3  |
| Thrown down by husband . . . . .            | 1  |
| Head injuries . . . . .                     | 8  |
| Straining at stool . . . . .                | 2  |
| No history of cause . . . . .               | 20 |
|   | —  |
|   | 52 |

*Time of Attack.*—At night, in 30 cases; during the day, in 22 cases.

The fact that the large proportion of these attacks occur at night, is an interesting one. They were mostly hospital patients, and some were irresponsible; so, of course, their statements are to be taken with allowance. One woman said: "I awoke in fright, and in attempting to rise found I was unable to do so." It is probable, therefore, that the condition was dependent upon disturbed cerebral circulation connected with nightmare; nearly every one of these thirty patients found that they were paralyzed only when they awoke in the morning, and attempted to get out of bed. Exposure to the sun's rays, and the stoppage of any flux that is either normal or pathological, are often sufficient to produce an attack, and as an example of the latter hemorrhoidal bleeding may be mentioned.

Hemiplegia may be a result of variola; and the following case, in which epilepsy and hemiplegia dated from smallpox, possesses much interest. The paralysis was due undoubtedly to an epileptic seizure, during which some vessel was ruptured.

M. J. T., 35 years, born in New York; no occupation; entered the Epileptic and Paralytic Hospital Feb. 11, 1870. Mother died of consumption; sister had epilepsy. First fits appeared at the age of five years; came on about three months after the attack of smallpox; hemiplegia of the right side came on at the same time, she believes, as the epilepsy. Before the convulsions she had cramps in the paralyzed arm and hand, and a feeling of dizziness; the attacks occur most frequently in the day-time, three or four together, and recur once in three or four weeks. But shortly before her admission she had them nearly every day. Circumference of skull, 20 $\frac{3}{4}$  inches; antero-posterior measurement, 12 inches; transverse, 13 inches; memory good, mind rather weak; speech good, sight good, hearing fair with left ear; cannot hear with right ear, even when the watch is pressed against it. Sensibility to pinching and pricking appear entirely abolished on the right side from head to foot. Drags right leg in walking; has but little use of right arm and hand, the muscles of which have a tendency to spasmodic contraction; temperature somewhat diminished on right side; appetite fair; bowels rather costive. Menstruated at 13 years, and has been regular since.

Present condition, June 1, 1876:—

Memory appears to be very good; and the fits have decreased in severity and in number. Had but two attacks last month; none at night. Has hæmoptysis sometimes before the attack, and an aura of about a

minute's duration; flexor of muscles of right hand is contracted: thumb is turned again, so that its inner part touches the under part of the index finger; lastly, the whole hand is somewhat drawn up, and lies in her lap with the palmar surface up. When directed to put hand up to shoulder, it shakes right and left; this shaking is very violent, but only so when she makes voluntary movement. It is, however, entirely quiet while in her lap. Has the irregular hemiplegic gait; protrudes her tongue straight; eyesight good; hears perfectly well. There is facial paralysis (peripheral) on the side opposite the hemiplegia, but no ptosis.

As an illustration of a curious case of cerebral hemorrhage, Eulenburg<sup>1</sup> relates the case of a switch-tender who, during a heavy thunder storm, inserted an iron key in the lock of a switch signal. He was suddenly deprived of power, and fell to the ground. After an hour or two, when sufficiently revived by the rain, he dragged himself to a neighboring station. He was paralyzed on the left side.

**Morbid Anatomy and Pathology.**—A vessel impaired by disease, and subjected to even the normal blood pressure, will very soon suffer changes in its calibre, insignificant perhaps at first, but afterwards far more serious, but, when the blood pressure is abnormal, and a force is exerted which the resilient character of the vessels enables them to withstand in the healthy state; the weakened portion gives way, and the brain-substance in the neighborhood is subjected to dangerous pressure. The character of the loss of function depends very much upon the importance of the vessels and their areas of distribution. The middle cerebral artery is especially liable to rupture, being in direct communication with the left side of the heart; consequently, the corpus striatum, optic thalamus, and parts supplied by this artery, suffer injury. The other large vessels follow next, and may be affected in various parts of their course.

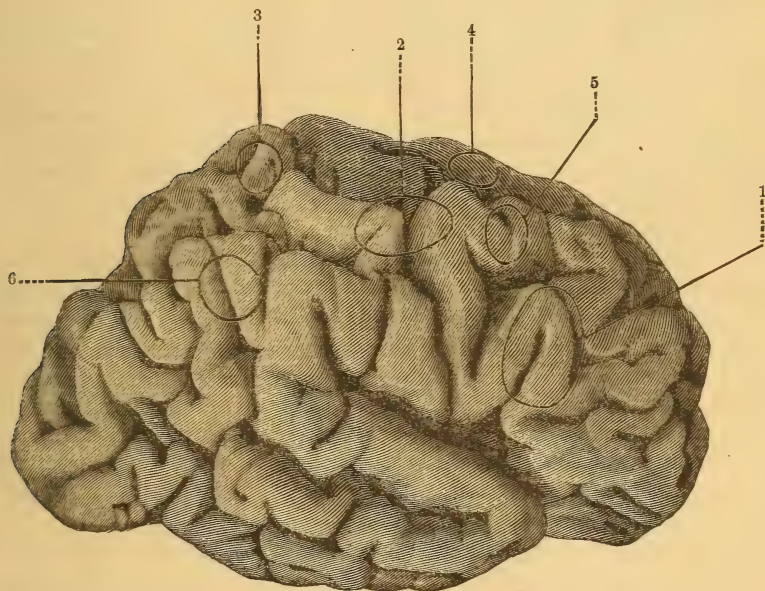
Such strides have been made in the study of cerebral anatomy and physiology during the past four or five years that it is necessary that the whole subject of nervous pathology should be viewed in a new light. New interest began with the researches of Jackson, Hitzig, Fritsch and Ferrier, and has since the discovery of the cortical centres been greatly increased by the valuable researches of Flechsig, Meynert, Huguenin, Charcot and a host of others. In the matter of central localization it behooves us to study the relations of the cortical psycho-motor centres and the so-called *pyramidal tract* comprising the descending fibres which run between the nuclei of the corpus striatum, and the optic thalamus, as the *internal capsule*, subsequently extending backwards and downwards as the peduncle (*crus*) and passing to the other side of the body, more or less fully in the pyramidal decussation.

The sensory ganglia, and the fibres passing from thence downwards, and the connection of the bulb with the cerebrum, come in also for consideration. It will be only possible in this limited space to consider the anatomical relation and physiological functions of these parts so far as they concern the occurrence of lesions.

<sup>1</sup> Berliner Klin. Woch., April 26, 1875.

The cortex-cerebri has been found to be the seat of well limited centres, which when subjected to irritation from disease or mechanical injury, lose the function of localized sensory and motor innervating power. The gray matter of the cortical motor region is found to be peculiarly rich in large giant cells such as are met with in the anterior gray cornuæ of the spinal cord, and by some authors are supposed to be identical with the latter. The more important of these centres are *motor*, and have been more or less appropriately called *psycho-motor centres*, and those of greatest significance are to be found upon either side

Fig. 16.



Cortical Centres. (Morel.)

of the Rolandic fissure in the ascending parietal and frontal convolutions, and preside for the most part over the movements of the face and its parts and the limbs of the opposite side of the body. There are more posteriorly other centres which have a sensory function. At the angular gyrus (*pli Courbè*), for example, a visual centre is found which fills a prominent office in the regulation of visual correction, while other limited regions exist which undoubtedly play an important part as centres for the sense of audition, taste and smell.

The excellent plate (Fig. 16) from Morel's Atlas will enable the reader to appreciate the action of the cortical centres. It is based upon the investigations of Ferrier.

1. *Speech centre of Broca.* Posterior part of third frontal convolution.
2. *Centre for the movements of the upper extremities*, situated on the



ascending frontal and parietal convolution circle (over the middle of the fissure of Rolando).

3. *Centre for the movements of the lower extremities.* Situated at superior extremity of ascending parietal convolution.

4. *Centre for movements of head and neck.* Over posterior extremity, or foot of superior frontal convolution.

5. *Centre for movements of lips.* Posterior extremity, or foot of the middle frontal convolution.

6. *Centre for movements of eyes.* Angular gyrus of parietal lobe.

These are in the main the important psycho-motor centres, although they are capable of modification, and I would refer the reader for further details to Ferrier's admirable book.<sup>1</sup>

The sensory centres, though more difficult to define, have occasionally been found to be the seat of disease, lesions being connected with limited loss of function. The centre of vision may be located in the supra-marginal lobule and angular gyrus in proximity to that centre concerned in the movements of the eyes, though it should not be confounded with an anterior centre situated upon the superior and middle frontal convolutions, which control lateral movement of the eyes and dilation of pupils.

The centre for *hearing* is located in the superior temporo-sphenoidal convolution. The centre for *smell* has been found by Ferrier in the subiculum cornua Ammonis, and irritation of this region is associated with some closure of the nostrils. The centre of *taste* is supposed by this author to be located in close proximity to the last mentioned centre. Many hundred observations have been collected by Charcot and Landouzy, Pitres, Seguin and a host of foreign and American observers, and most of them have a bearing confirmatory upon this theory, although it must be confessed that the large majority of collected cases present multiple or extensive lesions, which too often cloud the diagnosis. The published cases prove in several ways, and first that cortical alterations in places found by experiment not to be the seat of psycho-motor centres are not followed by hemiplegia, and this is shown by the cases of Pitres.<sup>2</sup> Two cases are presented by Pitres, one of softening of the inferior parietal lobule and sphenoidal convolutions, and the other of abscesses of the occipital lobe without hemiplegia. While other cases brought forward by him show the connection of hemiplegia with cortical softening of the ascending parietal convolution on one side, and aphasia with destruction of the third frontal convolution.

In cases where autopsies have been made it has been found that a degeneration of the motor fibres passing from this area of cortical centres had commonly taken place, and that such "secondary degeneration" had extended down into the cord involving certain parts of the lateral columns, to be alluded to hereafter, and this secondary trouble was

<sup>1</sup> The Functions of the Brain. London, 1876.

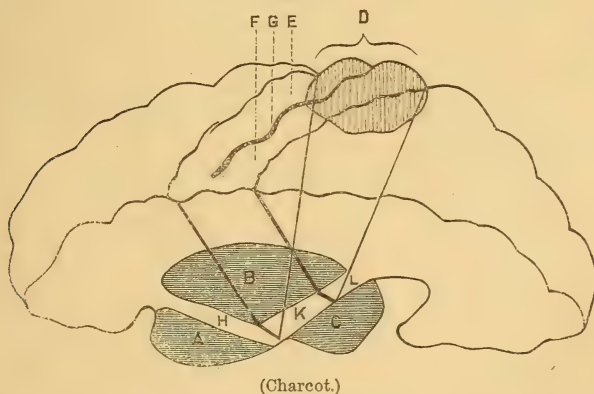
<sup>2</sup> Progres Medicales, August 7, 1880, and Revue Mensuelle.

found in some cases disconnected from any special lesion of the so-called motor ganglia, at the base of the brain, proving beyond doubt that the cortical psycho motor zone was that primarily affected.

The disturbances of motility observed in connection with such cortical degeneration have been found to be of two kinds, spasm and paralysis existing together or apart, the latter being but an extended stage of the former; and the interesting series of cases originally brought forward by Hughlings Jackson, who may be said to be the father of central localization, give to the matter an importance it really never has had accorded to it. It is the opinion of both Jackson and Brown-Séquard, both of whom have never been inclined to look upon the subject in anything like a narrow way, that the psycho-motor centres are not confined alone to the cortex, but exist throughout the brain as a complex system.

My own experience has led me to adopt this view, especially as I have seen cases in which the cortical centres of Ferrier were involved and in which the only disturbances of motility were hyperkinetic, such as localized spasms; and it would seem to me that the destruction of the cortical centres resulted more often in an interruption of inhibitory control than in intrinsic and primary abolition of motor power. There are numerous cases of cortical epilepsy in which no paralysis occurs, although the limitation of spasm to the member innervated by its particular cortical centre should always suggest the diagnosis. The occurrence of spasm in a monoplegic limb, that is to say a limb the seat of paralysis other parts being unaffected, is pretty certain to bear evidence of degeneration of a particular convolution.

Fig. 17.



When a large extent of cortical territory is destroyed we find a peculiar and extensive degeneration, which takes a well-defined downward course, as may be seen from reference to Charcot's admirable plate (Fig. 17). The zone which includes the psycho-motor centres above, and the inferior motor tracts, may be shown by a vertical cut which separates the hemispheres. A. represents the caudate nucleus;

B, the lenticular nucleus; C, the *optic thalamus*, while between them passes the collection of fibres known as the *internal capsule*. The relation of the nervous tracts with the convolutions above and the basal ganglia below is also shown in the diagram. D represents the paracentral lobe, which has been found to be the most important psychomotor region; E, the ascending frontal convolution; F, the ascending parietal; G, the fissure of Rolando. The various parts of the internal capsule are represented by H, K, and L. H represents the internal capsule; K, the "pyramidal" region of the posterior segments of the internal capsule, and L the part concerned in sensation. The anatomical arrangement of the internal capsule may be diagrammatically represented by the tract of white represented by the letters H and K in the above diagram. It will be noticed that these tracts unite at an obtuse angle, which latter by the Germans and French is known as the "knee of the internal capsule." The anterior segment of this collection of fibres contains those which are essentially motor, while the posterior are sensory. The knee contains fibres which terminate in the bulb and have a connection with some of the great nerves of the medulla concerned in the voluntary innervation of the tongue and other parts of the face.

In the diagnosis of cerebral disease it is well that we should bear in mind the relation of cerebral ganglia and their commissural connections, and a transverse section of the brain, when studied microscopically and otherwise, will enable us to see that not only are the two hemispheres connected together, but the various gray segments are brought into relation by different sets of fibres which may be briefly enumerated as follows: Fibres which connect the optic thalamus and the lenticular nucleus and the caudate nucleus with the periphery of the brain; fibres connecting the lenticular nucleus with the gray matter of the sphenoidal lobe. These internal intercommunicating fibres form a system by themselves, while a second set of fibres having a direct course, (peduncular fibres) serve for the direct reception and transmission of sensorial impressions and motor impulses.

After the fibres of the internal capsule reach a lower and more posterior level they unite in the peduncle, which, according to Brissaud and others, contains four sets of fibres, each having a well defined office and corresponding with the arrangement in the internal capsule. They are as follows: 1. A posterior bundle, the office of which is the conduction of sensory impressions. 2. A bundle composed of fibres especially engaged in the motor innervation of the trunk and limbs. 3. A small bundle of fibres connected with the angle (genou) of the internal capsule, and which contain motor fibres connected with the bulb<sup>1</sup> and are concerned in voluntary movements of the face, and tongue. 4. An internal bundle of fibres going to the bulb.

Evidences of secondary degeneration, after certain cerebral lesions in-

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<sup>1</sup> Loc. cit.



volving the motor track are best seen in the inner and middle thirds of the peduncle and sometimes occupy a pyramidal character the base being anteriorly.

The course of the motor fibres has been studied most fully by Flechsig in the embryo, and he has materially overturned the old views—notably those of Brown-Séquard in regard to the total decussation of fibres in the pyramids. Flechsig has found that the extent of decussation is very variable, and that in the great number of cases there is by no means total decussation. This will explain the possibility of hemiplegia upon the same side as the cerebral lesion in individuals in whom the pyramidal decussation is imperfect.

The study of sensory disturbances following brain lesions has not kept pace with that of the localization of motor troubles. Certain facts have been clearly brought forward, however, and the most important of these is that injury of the posterior segments of the internal capsule is productive of hemianæsthesia. Veyssiere<sup>1</sup> was the first to make this clear, and Charcot, Ferrier and others have since proved the connection of such unilateral anæsthesia with loss of smell and vision upon the same side. Injury of the convolutions about the fissure of Rolando has not been so far found to be followed by general anæsthesia, although according to Ferrier the occipital convolutions seem to some extent to possess sensorial functions. The optic thalamus has undoubtedly much to do with sensory innervation, and Friedrich and Charcot have both found that hemorrhage or tumor in regions adjacent to the posterior part of this organ produced anæsthesia, and in certain cases of epilepsy, with peculiar sensory auræ. Hammond has regarded the optic thalamus as the seat of the lesion.

The blood supply of the brain is derived from two systems of vessels, a basal or *central*, and a cortical or *external*.

It has been proved by Duret and others that there is no distal connection between these two, and that the central arteries as a rule supply but a limited territory. The importance of the central arteries, which are much larger than those supplying nutrition to the cortical gray matter, is derived from the fact that in rupture or disease much more profound and sudden symptoms occur than when the others are affected, because of the existence of anastomoses in the latter. Charcot alludes to several facts which in this connection should be borne in mind in the localization of symptoms. 1. Vascular lesions upon the surface of the brain and hemorrhages as a consequence do not occur so often as in the substance of the brain, for the reason that the cortical vessels are protected in their course by their dura mater and other coverings, that they are smaller, and are not subjected to so much pressure as those of the central system.

2. Proximity of the arteries of the central system to the heart—their simple arrangement and liability to sudden pressure predisposes to acci-

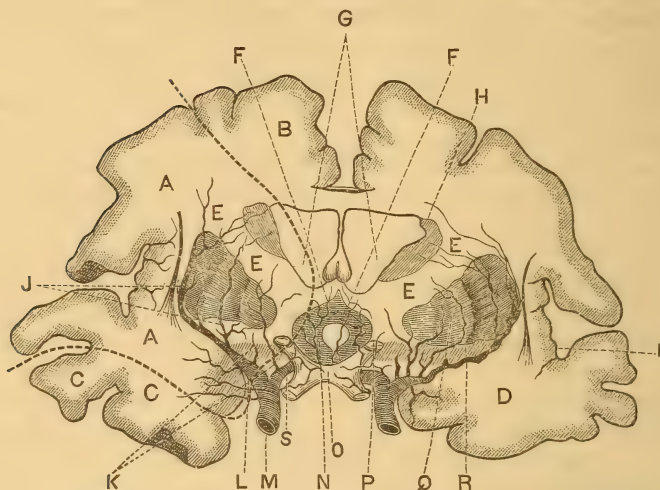
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<sup>1</sup> Recherches Clinique et Experimentales, sur l'hémianæsthesie. Paris, 1874.

dents in deeper parts, and for this reason central or deep hemorrhages are serious.

A reference to Fig. 18 will enable the reader to appreciate the vessels

FIG. 18.



(CHARCOT.)

Fig. 18., (Charcot). Central vascular supply. A. Territory of Sylvian artery. B. Territory of anterior cerebral artery. C. Territory of posterior cerebral artery. D. External wall. E E E E. Internal capsule. F. Walls of Trigonal arches. G. Lateral ventricle. H. Caudate nucleus. I. Island of Reil. J. External arteries of corpora striata. L. Sylvian artery. M. Internal carotid. N. Gray substance of third ventricle. O. Optic chiasm. P. Section of optic nerve. Q. Lenticular nucleus. R. External capsule. S. Anterior cerebral artery. Vascular areas are indicated by dotted lines.

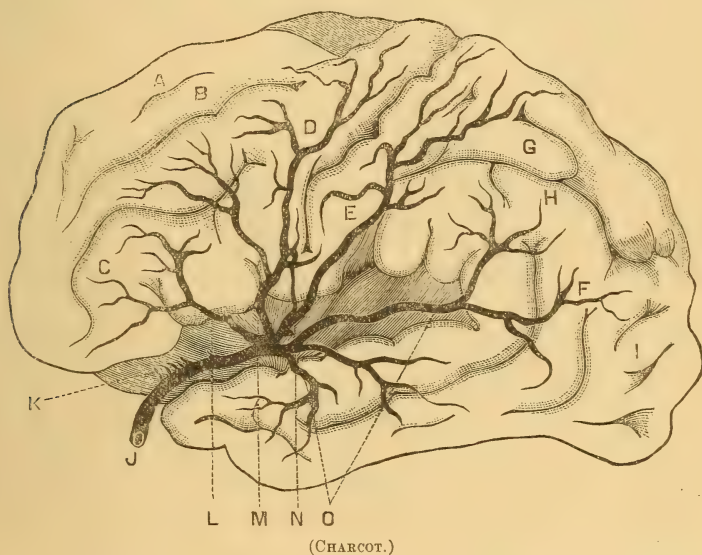
concerned in the supply of the central ganglia. The Sylvian or middle cerebral artery is the most important of these, and it will be found that when it leaves the internal carotid it sends up central branches to supply a part of the caudate nucleus, the entire lenticular nucleus, the internal capsule and a part of the optic thalamus. It will be seen by the dotted lines that nearly two-thirds of the hemisphere is supplied by this important vessel and its central and cortical branches. The posterior cerebral artery furnishes nourishment to the parts of the optic thalamus not supplied by the Sylvian—namely, the external and posterior parts. It also supplies the tubercula quadrigemina and the crura cerebri. The anterior cerebral artery is concerned only in the supply of a small part of the caudate nucleus.

Fig. 19 shows the course of the middle cerebral artery which sends off branches to supply the cortical portions of the brain after it fulfils an equally important office in supplying, at the base, central vessels to the ganglia.

The cortical branches of this vessel are quite large, and are four in

number. These severally supply the frontal, parietal, and sphenoidal convolutions. The island of Reil is supplied by a large branch which leaves the main artery when it divides into the large terminal branches. The four vessels alluded to, break up into smaller or *secondary* arteries at higher points, such secondary arteries supplying a small track of convolution. There are still "tertiary branchlets" which anastomose with each other forming arborescent ramifications—though Duret does not agree with Charcot and others regarding this fact.

FIG. 19.



Cortical branches of Sylvian artery. A B C. FRONTAL CONVOLUTIONS. D. ASCENDING FRONTAL CONVOLUTION. E. ASCENDING PARIETAL CONVOLUTION. H. INFRA-PARIETAL CONVOLUTION. G. SUPRA-PARIETAL LOBULE. I. OCCIPITAL LOBE. J. TRUNK OF SYLVIAN ARTERY. K. Perforating branches of central gray ganglia. L. Ext. and superior frontal branches. M. Ascending frontal artery N. Ascending parietal artery. O. Parieto-sphenoidal and sphenoidal arteries.

Upon the surface of the convolutions we find *nutrient arteries* of small size and capillary character, which are branches of the "tertiary branchlets." These arteries enter the cortex at a right angle with its external surface and are called *long* and *short*, with reference to their extent of penetration. The *long* or "medullary" arteries, are terminal vessels of the tertiary branchlets and pass perpendicularly into the gray cortex and white substance, *but have no connection with the cerebral arteries below*, while the *short* cortical or nutrient arteries, which also come from the tertiary branchlets or ramifications, rarely extend deeper than the cortical gray matter. The only difference in the character of the two forms of nutrient arteries, for they have a common origin, is that they extend to different distances from the cortical periphery, and while one supplies chiefly one form of nervous matter, (the white) the other nourishes the



gray. It will be found that a sort of arborization or net-work is found in the gray matter, which depends chiefly upon communicating arteries from the *short* vessels with an occasional reinforcement from the *long*, and also that the terminal branches of the large trunks are entirely distinct from those arising from a lower level, and which enter the brain at a basal point to become central arteries.

Other cortical parts of the brain are supplied chiefly by branches of the anterior cerebral, and posterior cerebral arteries.

The pathological course of cerebral hemorrhage is the following: 1. The stage of preparation, during which the arteries undergo the changes already spoken of. 2. The operation of an exciting cause, the rupture of the vessel, the injury of the nervous substance, and the formation of the clot. 3. Death; absorption, or limitation.

Bouchard<sup>1</sup> and Charcot both affirm that cerebral hemorrhage is always dependent upon a peculiar kind of disease of the vessels. This diseased condition consists of a studding over with minute aneurismal dilatations which have been called by them "miliary aneurisms." These arise from a primary degeneration of the outer coat of the vessel, secondarily sclerosis, and finally atrophy, of the muscular coat and dilatation. Of sixty-five cases of cerebral hemorrhage, they found miliary aneurism in every instance. Both of these authors consider the vascular change to be different from that of atheroma, which begins in the inner coat. These appearances are confined to the brain, and exist where there is no evidence of atheroma to be found in any other part of the body. Notwithstanding the fact that these views are endorsed by such men as Meynert, Bastian, and others, there are many observers who consider miliary aneurisms to be due only to careless manipulation, or to be identical with the "hyaline degeneration" of Gull and Sutton which is found in other localities.

Dr. Barlow<sup>2</sup> has presented a case which fully demonstrates that cerebral embolism may produce a condition of the vessels which leads to the formation of aneurisms, first causing local arteritis and weakening of the wall of the vessel. In this case (that of a boy aged ten years) there was right and afterwards left hemiplegia, and aortic regurgitation. The autopsy revealed "cortical softening on each side of the lower part of the ascending frontal and the posterior parts of the second and third frontal convolutions. The clue to this condition was found in the middle cerebral arteries. On both sides these vessels were diseased at the spot where the fine branches were given off over the island of Reil for the supply of the cortex. Of these branches on both sides, the one supplying Broca's convolution and the one supplying the ascending frontal were also diseased. There was no aneurism to be discovered anywhere, but the walls of these vessels presented many small calcified nodules obvious to touch and sight." This calcification was not noticed in any other

<sup>1</sup> Archives de Physiol., 1868.

<sup>2</sup> Brit. Med. Journal, April 7, 1877, p. 372.

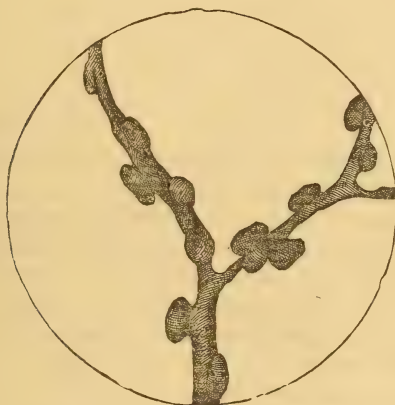
vessel in the body, and emboli had lodged in the spleen and kidneys. In Goodhart's cases actual aneurism had followed the embolism, and Dr. Barlow's case demonstrates that there is a primary weakening.

Durand-Fardel<sup>1</sup> found that of 32 cases the arteries were only healthy in 9 cases, while in 21 they were thickened, and in 2 ossified.

Andral<sup>2</sup> found that of 32 cases the arteries were apparently healthy in but 4.

These miliary aneurisms have been said to be due to "periarteritis," but it cannot be denied that a large proportion of cases of renal and heart disease produce modifications in blood pressure, which would account for the rupture of the vessel without any primary inflammatory condition.

Fig 20.



Miliary Aneurisms.

I have repeatedly seen miliary aneurisms, and must confess that they appeared to depend upon some organic change which extended over a considerable space of time.

Zenker differs from Charcot and Bouchard, and considers the internal coat to be that which is first attacked. When miliary aneurism exists, it is generally in conjunction with either gout, cancer, tubercule, leucocythemia, or other conditions, when leucocytes may pass into the cerebral vessels in large number. In old drunkards and general paralytics this vascular change is not an uncommon one. In regard to atheroma there have been many cases brought forward where this appearance was so constant as to gain recognition as one of the chief factors of the cerebral hemorrhage. An atheromatous artery contains deposits of a firm, semi-fatty nature, between its inner and middle coats. At an advanced stage the deposit is more calcareous and hard, and the artery may be sometimes easily broken in two. Occasionally the deposit between the coats, by distension considera-

<sup>1</sup> *Traité clinique et pratique des Maladies des Vieillards*, Paris, 1854, p. 223.

<sup>2</sup> *Clinique Med.*, vol. v.

bly narrows the calibre of the vessel, and in this way forms occlusion at one point while at a weaker one hemorrhage takes place. The veins and capillaries are not so often involved as the arteries. In regard to the seat of cerebral hemorrhages, we find from a table prepared by Rosenthal.<sup>1</sup>

|   | Times. |
|---|--------|
| In the corpus striatum alone . . . . .  | 32     |
| “ nucleus lentiformis alone . . . . .   | 20     |
| “ both these ganglia combined . . . . .   | 8      |
| “ corpora striatum and optic thalamus . . . . .   | 7      |
| “ cent. nucleus and other parts (centrum semiovale, occipital lobe,<br>island of Reil, pons and cerebellum) . . . . . | 6      |
| “ optic thalamus alone . . . . .  | 20     |
| “ “ “ and corp. striat. of both sides (recent hemorrhages<br>and old cicatrices.) . . . . .                           | 2      |
| “ “ thalamus and lent. nucleus of both sides . . . . .  | 3      |
| “ centrum semiovale . . . . .   | 3      |
| “ parietal lobe. . . . .  | 2      |
| Total . . . . .   | 103    |

It may be stated that large portions of both hemispheres are destroyed without serious symptoms; but when we approach the base the danger is increased, and if the third frontal convolution be the seat, we find a very decided and serious result, which is aphasia. The majority of hemorrhages are in or about the optic thalami and the corpora striata, together or singly, and if they be extensive the ventricles will be filled. If the hemorrhage be great, pressure may be made on the opposite side, or the blood may find its way into other localities. In the anterior lobes the effusion is generally circumscribed, but from this site it may find escape into the lateral ventricles. In the ganglia and important parts at the base, the hemorrhage is generally small, but is all the more serious because of the importance of the parts it destroys. This is the case in the corpora striata. In the pons and medulla any considerable extravasation is followed by death or serious trouble. The shape of the cavity is variable, but in the gray matter it is circumscribed, and in the white it is irregular and elongated.

Parrot<sup>2</sup> reports 34 cases of cerebral hemorrhage in new-born children. In these the clot was found at the inferior part of the brain; sometimes on the right side, but more generally on both sides.

Should the patient survive the apoplectic attack, and die subsequently of some other disease, the cerebral clot will probably prove to be well organized, hard, and separated from the brain-tissue in the vicinity by a sclerosed mass. The immediate changes are the following: At the end of a few days the serum is absorbed, leaving the solid portion as a gelatinous mass; finally the clot contracts, becomes yellow, and assumes the appear-

<sup>1</sup> A clinical treatise on the diseases of the nervous system, translated by L. Putzel N. Y., 1879, p. 38.

<sup>2</sup> Arch. de Tocologie, 1875.



ance I have alluded to. It is rare that an old clot is completely absorbed, but it is found encysted and firm, and, perhaps, has produced some softening. It is not uncommon to find more than one clot in a patient who has had several hemorrhages. There may be a cyst filled with thickened blood, which is indicative of an effusion of recent occurrence, and there may be others of smaller size, in different stages of resolution. Small aneurismal dilatations are also found, while local patches of softening, or cysts filled with clear serum, are not rarely present at the same time. Much has been said about the relation of decubitus to brain lesions; however, there does not seem to be any special connection between disease of certain parts of the brain and the causation of bad sores, though Joffroy<sup>1</sup> has reported three cases in which acute decubitus was found with lesions of the occipital lobe and optic thalamus upon the opposite side. Broadbent, Dusaussay, Leloir and others have, however, presented a number of cases in which other parts of the brain were affected.

A common form of hemorrhage is meningeal. Goodhart<sup>2</sup> has written an exhaustive paper upon this subject, in which 49 cases are given, proving most conclusively its connection with diseased kidney and hypertrophied heart. Of these 49 cases, 30 were due to renal disease, and six had uncomplicated heart trouble. When the hemorrhage takes place above the arachnoid, we are assured by Mr. Prescott Hewitt<sup>3</sup> that the blood very rarely gravitates to the base; but when the hemorrhage is sub-arachnoid, the blood may find its way below, thus making the condition a most serious one. After death a peri-cortical collection of blood will be found; which is extensive over the base, and probably produces death by pressure upon the pons and medulla. (See Chronic-Pachymeningitis with Hematoma.)

**Diagnosis.**—Coincident with the occurrence of the hemorrhage, symptoms will be presented which may enable us to localize with some degree of accuracy the position of the clot, its extent, and character, and the following statements are based upon the observations of Bastian, Wilks, and others: *A lesion in or about the corpus striatum* will be followed by hemiplegia of the opposite side. The temperature being higher in the paralyzed limbs than in the others; the eyeballs will deviate towards the side of the lesion; and the tongue, when protruded, will point to the hemiplegic side. The face is paralyzed on the same side as the arm and leg. *A lesion in or about the optic thalamus* will present the same phenomena, only that the temperature is higher in the paralyzed limb than in the preceding form. *A lesion in one crus* is followed by very much the same symptoms. If the under and inner part be affected, we find *cross paralysis*, the face being paralyzed on the side of the lesion, while the extremities are paralyzed on the other side of the body. Hemianæsthesia is quite marked; and the third and seventh nerves are paralyzed, so that ptosis and profound

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<sup>1</sup> Archives de Medicine, Jan. 1876.

<sup>2</sup> Guy's Hosp. Rep., vol. xxi. p. 131.

<sup>3</sup> Holmes's System of Surgery, 1870.

facial paralysis result. *A lesion in one lateral half of the pons* is followed by hemiplegia of the opposite side, profound coma, deviation of the eyes away from the side of the lesion, facial paralysis on the side of the lesion, lowered temperature in the non-paralyzed limbs, paralysis of the muscles of deglutition, and anæsthesia or hyperæsthesia of parts supplied by the fifth nerve. *A lesion of the upper half of the lateral region of the pons* will be expressed by pretty much all of the symptoms which follow the last mentioned lesion, except that the facial paralysis will be on the side opposite the lesion. A feature of all forms of lesions in the pons is the very decided character of the facial paralysis; and if there be extension of the lesion, there may be double facial paralysis, with hemiplegia of the body. *A lesion in the posterior part of the pons*, beside the symptoms just alluded to, will produce paralysis of the fifth, sixth, and seventh nerves on the side of the lesion; or, according to Brown-Séquard, it may sometimes produce cross-paralysis. *A lesion in the centre of the pons* is followed by double paralysis, deep coma, marked contraction of pupils (while in the other forms one pupil may be contracted on the side of the lesion), lowered temperature on both sides, with ultimate rise and but slight loss of sensation. Liouville<sup>1</sup> reports a case of hemorrhage into the pons, in which sugar was found in the urine. This he considers to be an ever-present symptom of disease in the lower part of the pons, but never a feature of disease of the upper part. A hemorrhage in the medulla is followed by paralysis of the cranial nerves on both sides, bilateral paralysis of the body, and, generally, rapid death. Extensive lesions may produce a combination of these phenomena, and diagnosis may sometimes be an extremely difficult matter. A patient under treatment with syphilitic disease of the brain, presents a combination of symptoms which are extremely interesting in a diagnostic sense.

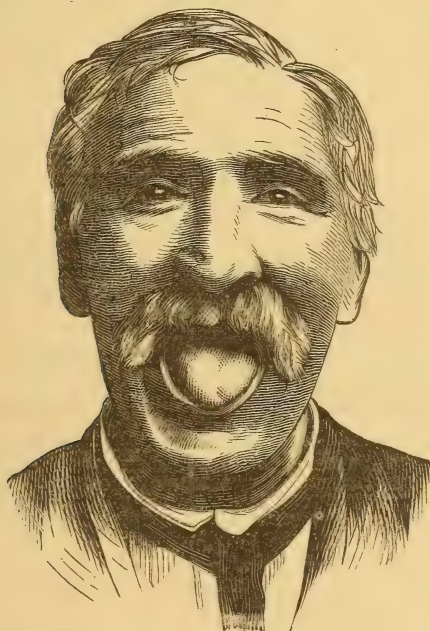
Wm. McG., aged 58 years, when about 21 years of age, had a primary chancre upon the dorsum of the penis, followed some months afterwards by secondary symptoms. After a few years all traces of syphilitic trouble seemed to have disappeared, as he enjoyed extraordinary good health. He has led for the last twelve or fourteen years a very intemperate life, and has regularly "gone upon sprees." Twenty-six months ago, after an attack of facial neuralgia, which was evidently specific, he became hemiplegic during one of his drinking bouts, but does not remember any of the circumstances immediately connected with the apoplexy. When he became sober he found that the left side was paralyzed, but the loss of power could not have been very great, for he was able to walk in a few days. About a year ago the *right* side of the face became anæsthetic, and he began to lose the sense of taste on the *left* side; at the same time he found it difficult to arrange the food for mastication, and his power of articulation became embarrassed.

**PRESENT CONDITION.**—*Eyes.* Pupils of the same size, and not abnormal; respond well to light; no ptosis, nor disturbance of vision; no retinal change. *Face.*—No impairment of buccal muscles, nor of superficial facial muscles, except slight contraction of those of right side when he

<sup>1</sup> Gazette des Hôpitaux, Feb. 8, 1873.

opens his mouth. When this is done, the orifice is unsymmetrical. Anosmia marked, taste impaired to slight degree. Warm substances produce an impression on sound side of tongue, but not on the other. Left side of the palate paralyzed, and lower than the other. Left side of tongue atrophied, presenting the appearance depicted in Fig. 21; and when protruded the tip points to the right side, no apparent tactile loss of sensation as determined by the æsthesiometer. Saliva is secreted in large quantities, and

Fig. 21.



Multiple Lesion with Tongue Atrophy.

constantly drips from the angles of the mouth when he talks. Sensation of right side of face impaired; feels points only when separated 3 mm. on other side  $1\frac{1}{2}$ ; some difficulty of speech, especially with the letter r, pronouncing "righteous" "eightshus;" the left leg he drags slightly when he walks. Six months ago he slept upon his arm when drunk, and thereby added to his other troubles a decubitus paralysis; slight loss of power in both arms.

In this case there were evidently two lesions—one in the medulla, and the other on the right side of the brain—one hemorrhagic, the other of slow growth.

We are to diagnose the symptoms of cerebral hemorrhage in its different stages from those of the following diseases: *Actual attack* from uræmia, drunkenness, opium poisoning, tumor, epilepsy, compression or concussion from injury, embolism, and thrombosis. There are certain general appearances which symptomatize the *uræmic* condition, and can hardly be mistaken; the skin is waxy and œdematous, the eyelids are puffed, and



the legs and feet swollen; but, as Bastian suggests, it does not always follow, when we find these appearances in an individual over thirty years of age, that the coma is always purely of an uræmic character, and that there may not be a complicating hemorrhage. The urine, when drawn, is found to contain albumen, but this symptom by itself is insufficient to settle the question. Uræmic coma is generally of gradual appearance, though Hughlings Jackson calls attention to a form which has a rapid onset, with convulsions; but, on the whole, such sudden appearance is more suggestive of cerebral hemorrhage. It is nearly always preceded by prodromata for several days. The patient is stupid, and inclined to somnolence, and has headache. Bourneville has ascertained that the temperature rapidly sinks when the coma begins, to a point very much lower than it does in cerebral hemorrhage, and continues depressed during the condition, while the converse is true in the other affection. Convulsions are much more prominent and constant features of uræmic coma than they are of cerebral hemorrhage; and, beside, there is no paralysis. Numerous other indications will serve to make the diagnosis clear in this respect. The coma is not deep, and it is possible to arouse the patient, and there is great hyperkinesis, there being a tendency to muscular spasm and rigidity which is not unilateral. The character of the respiration differs from that of cerebral hemorrhage, the stertor being more superficial. From *drunkenness* the diagnosis is not always so easily made, the two conditions sometimes coexisting, and it may be necessary to delay until the effect of the alcohol has passed away, before we can determine our patient's true condition. The odor of liquor, the circumstances under which he was found, and his imperfect loss of consciousness, are sufficient to excite suspicion. If he vomits, we may chemically test the substances thrown up, or examine the urine. Anstie gives a delicate test which may be employed. If even only one drop of the urine of the patient who has taken a toxic dose of alcohol be added to fifteen minims of a solution of one part of bichromate of potash in three hundred parts of strong sulphuric acid, the mixture will turn to an emerald green. With a larger quantity this test will be much more certain. The articulation of an intoxicated person when aroused is so peculiar and so interrupted by hiccough that there need be no chance for mistake in this respect. *Narcotic poisoning* may resemble somewhat the symptoms indicating cerebral hemorrhage. Like alcoholic coma, its advent is gradual, and there are convulsions, while the face is dusky, but the patient may be generally aroused. Much stress has been laid upon the condition of the pupil in opium poisoning as a diagnostic sign; but, as this symptom is indicative of hemorrhage in the pons, it loses some of its value. *Epileptic coma* can hardly be mistaken (should it be a stage of the actual epileptic attack) for that of cerebral hemorrhage. In the former there is a history of convulsions; the stupor lasts but for an hour or two at the most; the temperature is elevated; and there is sometimes an escape of bloody froth from the mouth. The previous history of the patient should set all other doubts at rest. *Compression or*

*concussion* from head injuries may be mistaken for the condition under consideration. In the former there may be a subarachnoid effusion, which may give rise to many of the symptoms. The latter is usually of short duration, so far as symptoms are concerned. The skin is pale, the pupils dilated, and vomiting occurs at some time or other. It is always of decided importance that we should inquire into the nature and receipt of the injury; for, should it follow a fall while the patient is in a safe position, we may suspect that he has had a seizure of some kind, the injury being secondary to the attack.

The internal cause of the hemorrhage is always important, whether it be produced by an abscess, tumor, or other intracranial disease states; and these things are to be taken into account. The antecedent history of the patient, the presence of pain of a localized character, subsequent convulsion, loss of vision, aural disease, and kindred conditions should all be ascertained. *Serous apoplexy*, as it has been called, when an immense effusion of serum takes place either beneath the investing membrane, or in the ventricles, or throughout the brain substance, is usually of gradual origin, and dependent upon the collection of fluid which takes the place of atrophied brain substance or attenuated vessels.

**Prognosis.**—According to all observers it is an exceedingly difficult matter to make a prognosis with any certainty, especially an early one, and, consequently, it is of the utmost importance that every circumstance of the case should be taken into account and carefully considered before we give expression to any opinion. Certainty of prediction is made doubtful, by new complications, and fresh dangers that are likely to arise. There are several questions that are to be answered, and the first of these concerns the fatality of the actual attack. The character of the coma, its depth and duration, the appearance of convulsions, abolition of reflex excitability, stertor, involuntary passage of urine and feces are to be regarded as indicative of an early fatal termination. If this condition be connected with unequal pupils, and double hemiplegia, the prognosis is, if anything, more unfavorable. Large hemorrhages into the ventricles, corpora striata, or into the crura or pons are then to be feared. The patient presenting these alarming symptoms dies usually in a very short time, say in from a few hours to two or three days, and there may be, perhaps, an aggravation of the symptoms towards the end as the result of fresh hemorrhage. If he survives the attack, what are the chances for the return of mental power? or, if not affected, will it subsequently become impaired? This depends very much upon the occurrence of inflammatory action about the clot, or whether there be uræmic trouble or softening. We may augur well for his chances if these conditions are absent, and if he lives for eight or ten days after the immediate attack. In regard to the speech disturbances: if there be simple ataxia, there is no reason to fear; if, however, any marked forgetfulness of words or genuine aphasia exists, the prognosis is less hopeful. This condition of affairs often exists for years without the slightest

improvement taking place. At first the mind is confused and dull, and, unless the hemorrhage is the result of softening or other degeneration, there is but little doubt that he will ultimately regain his mental activity. It is, however, well to qualify this statement by saying that in old people the tendency is the other way. Congenital apoplexies, or those occurring in early life, are apt to leave sequelæ of the most deplorable description, such as imbecility and kindred conditions. The return of muscular power and normal sensation is the most important question to be next considered, for much of the patient's future comfort depends upon the recovery of his lost power. Should the limbs remain paralyzed, or secondary neuritis take place, the consequence will be atrophy and contractures, such as I have described. It is, however, usual for recovery to begin in a few weeks, and in even a shorter time should the hemorrhage be unattended by loss of consciousness. The limb first to recover is the lower extremity. He is able after a short time to get out of bed and "hobble" about, or he may retain a certain degree of power from the first should the hemorrhage be slight. He is subsequently able to raise his hand to his head, and ultimately recovers entirely. But this improvement does not always occur, for during cerebritis, and secondary degeneration which may subsequently take place, a number of serious muscular distortions of a permanent character may ensue. A case illustrating this is the following:—

J. C. D., aged 53, born in Ireland; carman. Family history, mother died of old age; father died of renal disease. The patient in early life was very intemperate, and there are some evidences of syphilitic trouble, there being nodes, bald spots, and enlarged glands; but he denies any venereal disease. For three months previous to the attack (it occurred three years ago) he suffered from headache, dizziness, and other prodromal symptoms; none very marked, however. He went to bed one night feeling perfectly well, and awoke with "cramps," which affected his right leg; he called his wife, and attempted to get out of bed, when he found he was paralyzed. There was no speech trouble whatever. He was placed in bed, and remained there for three months, during which time he had violent headache in the occipital region.

*Present Condition.*—Hemiplegia of right side, sensibility slightly impaired, and no atrophy of either the arm or leg. When he stands there is slight rigidity of the inner ham-strings. The toes and the end of the foot are adducted; and when he walks, the foot is raised from the ground about one inch; the knee is rigid, and there is motion only at the hip-joint. The fingers of the right hand are in a condition of extreme flexion, and cannot be extended by ordinary force; but, when the hand is placed in hot water for some time, the rigidity is partially overcome. The thumb is not involved; but, when the distal phalanx was extended, it could be bent backwards some distance, and remained in this condition until it was restored by me. The hand is slightly flexed, and the forearm pronated and flexed on the arm, and the arm adducted to the body. No lateral movement is possible. There was an early history of neuritis, which came on a short time after the attack, with decided pain in the shoulder-joint, during which the patient applied blisters and mustard poultices. The dynamometer indicates 20, outer circle, with the right hand, and 80 with the left. There is no visible facial paralysis, but the



tongue points slightly to the right side. The surface of the paralyzed side is mottled and cold, and the nails are crenated and horny.

The facial paralysis is sometimes a grave and permanent condition, and is very serious, especially if there be ptosis. Should the paralysis involve the muscles of the pharynx, the tongue, or the buccal muscles, the prognosis is very bad, and these symptoms suggest that the hemorrhage has invaded the posterior basal parts of the brain, and perhaps the medulla. The organs of special sense are affected to a variable extent, and greatly modify the prognosis. If there be involvement of the optic-discs, retinal extravasations, or structural changes of the fundus, a grave character is given to the disease; while such symptoms as ptosis and diplopia, which depend upon paralysis of the third and sixth nerve, sometimes disappear after a time, though such disappearance may very slowly take place. The recurrence of apoplectic attacks is not uncommon, and if there be any special cachexia, they are to be dreaded. Syphilis and gout, as well as renal disease, are highly conducive to a return of the trouble; or advanced age is an important predisposing cause of cerebral hemorrhage. When we find a calcareous state of the arteries with cerebral hemorrhage, it is very probable that the other fluxions will follow. I remember a case in which a succession of hemorrhages occurred in the person of a middle-aged lady, the third of which proved fatal:—

N. G. A., aged 57. On the evening of February 3, 1873, I was called by Dr. Wm. H. Bennett to see the patient, whom I found in a state of coma. All of the characteristic appearances of a profuse cerebral effusion were manifested. The apoplectic seizure had taken place the day before, and she had continued in a comatose state until I saw her with Dr. Bennett. Her surface was cool, her breathing slow and stertorous, her pupils dilated, and cornea insensitive to the touch; while reflex excitability was entirely abolished, so that tickling of the soles was followed by no withdrawal of either limb. In this state she remained until the 8th of the month, during which time, and in fact until the time of her death, in November of the same year, it was necessary to draw her water nearly every day. At the end of the fifth day there was a slight return of consciousness, but entire inability to speak, the patient making a peculiar short sound when she wished to communicate with those about her. There was complete paralysis of the right side, but a faradic current readily produced muscular contractions. From this period until September 13th, there was steady improvement, and the family, as well as ourselves, were very hopeful. She recovered considerable power over the leg and arm, but was unable to get out of bed, although she was lifted from it and placed in an easy chair, where she remained contented for several hours of the day. She was now able to utter two or three words, and seemed to take a lively interest in all that went on about her. On the 13th of September, while lying in bed, she suddenly became comatose, and presented all the symptoms of a fresh hemorrhage. Her temperature, which had before ranged between  $98^{\circ}$  and  $101^{\circ}$ , now sank to  $96^{\circ}$ ; and her condition was so critical that I remained with her during the night of the 14th, when she slightly recovered, regaining her consciousness on the 17th; but there was complete loss of power. The temperature now rose to  $104^{\circ}$ , and she was restless and irritable. Her power

of expression had entirely disappeared, and she remained in this state until the 19th of November, when she died in her last apoplectic attack.

This patient, before her last illness, had suffered for some time from albuminuria, but her symptoms had been almost entirely relieved when her first cerebral hemorrhage took place. She was of spare build, her radial arteries were rigid, and the arcus senilis was visible to a limited extent.

This tendency to cerebral hemorrhage is sometimes seen in gouty subjects. A patient recently sent to me by Dr. William Lockwood, of Norwalk, Conn., had suffered for years from gouty trouble. Besides the pain, her joints presented gouty swellings, with chalky concretions. Within the past five years she has suffered from slight hemiplegia of both sides; on the right most severely. In this case it is probable that the rupture of a large vessel will some day carry her off.

**Treatment.**—Our treatment must be, *first*, preventive, *second*, for the attack, and *third*, for the amelioration of the resulting condition. If we have to deal with cachexias of different kinds, appropriate treatment is indicated. Should there be gouty trouble, albuminuria, or syphilis, these are to be met with alkalies, diuretics, and specific remedies, such as mercury and the iodides. If there be depraved general health, weak heart action, and general debility, we are to support our patient by quinine, stimulants, and nourishing food. Combinations of digitalis and iron are especially useful when there is low arterial tension, and rapid heart action. In speaking of cerebral congestion I alluded to the conditions which might favor an excessive flow of blood to the head, and advocated special forms of treatment. It is not necessary to repeat these indications, but I will simply refer to the value of the bromides given in doses of from 20 to 30 grains three times a day if there be any tendency to head fulness, while ergot administered in half-drachm doses two or three times during the 24 hours, and the abstraction of blood from behind the ears, may be resorted to, should there be a suspicion of immediate danger. The patient is to be kept perfectly quiet in a cool room, cold applications are to be made to the head, and his bowels should be emptied by some such cathartics as the compound jalap powder, senna, or Rochelle salts. Should we recognize the appearance of any prodromal symptoms, we must immediately inform the patient of the dangerous possibility, and enjoin upon him the necessity of regulating his mode of life, of breaking off bad habits, and using every means in his power to improve cutaneous circulation. The flesh-brush, cold, and sometimes Turkish baths, moderate outdoor exercise, and other agents which stimulate the surface capillaries and relieve internal congestion, should be as soon as possible resorted to. The patient's diet should be farinaceous, and the use of either strong drink or condiments is to be at once discontinued. He is to sleep in a cool room, and on no account wear tight neck gear. The feet are to be kept warm, and thick woollen stockings should be recommended. Violent exertion, especially forms requiring any fixation of the abdominal muscles or

straining, are also to be carefully guarded against. Should we be called to find the patient in the actual apoplectic state, another line of treatment must be followed out. If in this condition he is found lying in a comatose state upon the floor, he is to be lifted gently, carried to a bed, and well propped up by pillows, so that the head is elevated. The room should be kept cool and well ventilated, and cold applications are to be applied to his head, while his feet may be kept warm by contact with bottles filled with hot water. The room is to be darkened, and his collar and shirt collar band should be cut or ripped off, so that the flow of blood to and from the head shall be unembarrassed. It is essential to keep him perfectly quiet; so loud talking is to be forbidden, and officious friends kept away. In times gone by, it was customary always to bleed at this stage. I think experience has clearly proven how dangerous is such practice, for hemorrhage in the brain is very apt to be started afresh by any such measure. If, however, the pulse be full, strong, and bounding, the patient's face flushed, and his condition one of plethora, the abstraction of a few ounces of blood from behind the ears, with cold douches to the head and mustard plasters to the calves, will do much good. This condition may be so patent to the observer that, perhaps, in *rare* instances, and after *careful deliberation*, he may decide to abstract ten or twelve ounces from the arm. If we hear that he has been constipated for several days, a drop or two of croton oil or half a grain of elaterium may be given in a wafer, or applied to the tongue if he is unable to swallow; it is advisable to give the first remedy, however, if the patient is profoundly comatose. Should there be much cardiac excitement, no better medicines can be recommended than tincture of veratrum viride, or tincture of aconite; the former in doses of from 6 to 8 minims till the pulse force is decreased, and the latter in rather large doses, say from 4 to 6 minims at a time, and after an interval of four hours, another dose, if the pulse has not decreased in volume or frequency. The medical attendant should not forget to draw the patient's urine frequently. I have known a neglect of this precaution to be followed by pain and distress which the patient in his helplessness is unable to express; and I cannot impress too strongly upon the student the necessity of remembering this simple procedure. When consciousness returns we may continue the aconite if it is indicated, and perhaps combine it with small doses (say 10 grains) of the bromide of sodium every two hours. Active medication of any kind, however, is injudicious in the extreme; so it will not do to give large doses. Should there be a condition of prostration, a tablespoonful or two of milk punch may be given every few hours. The subsequent management of the case is sufficiently simple; continued quiet, a moderate quantity of food easy of digestion, and attention to the functions of the body are the three indications. He should not be allowed to get up to defecate, but the bed-pan may be placed beneath him. It may be found necessary to give an enema, which is better than the administration of purgatives by the mouth, and in this case the patient should not be allowed out of bed, even though he may seem bright and sufficiently strong. Cleanliness should be insisted upon, and



generally necessitates the faithful care of a responsible nurse; for, if the patient is not carefully washed, the irritation produced by alkaline urine and his loose evacuations may favor the development of bedsores. As a precautionary measure, the buttocks should be rubbed with salt and whiskey, or, what is still better, tannin and alcohol. Bedsores may occasionally form, and sometimes are unnoticed by the physician if he is not on the alert, until his nose or the nurse remind him of their existence, the patient either being unconscious of such trouble, or unable to inform the physician even if he is aware of their presence. The patient should be immediately put on a water bed, and the slough removed by poultices of flax-seed and charcoal which may be sprinkled with iodoform. At the end of the 8th or 9th day, should the tendency be to recovery, and the temperature normal, we are left with an ordinary case of hemiplegia. What is to be done next? If the attack has been a serious one and signalized by marked loss of consciousness, and if the secondary rise of temperature be high, it is not best to begin electrical treatment for fully a month or longer. If the muscles respond too quickly to electric stimulus, we are not to use this agent, but to wait for some days or weeks, when we may cautiously employ the faradic current to the muscles of the affected side. Large sponge-covered electrodes moistened in a salty solution should be employed, so that all the muscles may be subjected to the electric stimulus in turn. Electrization may be direct or indirect, the muscles being made to contract either when both sponges are applied to their bellies, or when one is placed in contact with the muscle and the other is applied over the motor nerve by which it is supplied. In certain cases faradization fails to do any good whatever, and this is especially the case when there is delay in the absorption of the clot or any cerebritis. Two cases illustrating the possible advantages of this form of treatment are the following:—

*Right Hemiplegia.*—O. S., aged 52, butler, came under my charge October 2d, 1872. He had been deprived of consciousness and power of motion a year before by a cerebral hemorrhage, and, after resuming the duties of his avocation some months afterwards, continued well till three months ago, when a second attack prostrated him; but, through the good treatment he received at Bellevue Hospital, he partially recovered the power of locomotion. When he came to me for treatment there was complete hemiplegia of the left side. There was no peculiarity in his gait, beyond a very slight dragging. The arm was slightly atrophied, and the amount of power exerted by a forcible grasp of the dynamometer was indicated by 15° of the lesser circle. He could not button his clothes, nor lift his arm above his head. There was no difficulty in speech, except it might be embarrassment in speaking the words containing the letters “b” and “p,” when the labial muscles were required.

Electric irritability in the arm was slightly exaggerated. After giving him a simple prescription for his constipation, I dismissed him.

In three weeks afterward he returned in very much the same condition. I then systematically applied the galvanic current to the head, and the faradic to the limbs. The improvement was marked and imme-

diate. The muscles lost their atrophic state, and became firmer and larger. The patient was able to perform many actions with his hands not possible before this treatment. Faradization to the lips and cheek has effectually overcome the facial paralysis, and he now speaks distinctly.

*Cerebral Softening ; Right Hemiplegia.*—H. Walker, aged 62, Germany, canal-boat captain, presented himself for treatment in December with a well-marked right hemiplegia. He had been injured some time before while on the deck of his canal-boat, and then hit upon the head. He was senseless for some days, but recovered, with severe cerebral disturbance, which, from his wife's statement, must have been inflammation of the cerebral substance.

He left his bed after some weeks, with persistent pain in the head, aphasia, trembling, and a heavy feeling of the lower limbs. His memory and other mental faculties became obscured, and there was an uneasy expression of the eyes. About a year after the receipt of his original injury, while working one day in the sun, he had an apoplectic fit.

After remaining in bed some time, muscular power and cutaneous sensibility slowly came back. He was able to walk with difficulty; his speech was indistinct; the muscles of both the leg and arm were greatly atrophied; and I determined to use faradism.

The constant use of the *very mild* current for several weeks brought back, to some degree, the original contour of the paralyzed muscles. He was able to progress with a cane, but his speech remained imperfect. During the treatment he had repeated premonitory signs of a new attack. Faradism was resorted to to prevent atrophy, but its good effects were only temporary, as there is still softening.

In connection with this treatment we may give at the same time either iodide of potassium, strychnine, or ergot.

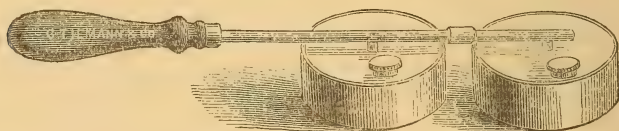
*Iodide of Potassium.*—Should there be a syphilitic history, I think we may begin at once with this remedy. If there be no such dyscrasia, I do not approve of the remedy at any time. It is administered very often with the idea of producing absorption of the clot, and is recommended by many writers. My limited experience has convinced me that its virtues have been very much overestimated. I have found that in many cases the patient's tendency to recovery was hastened more by rest, good food, and fresh air, than by any other form of medication. It is perhaps of value in old cases.

*Phosphorus.*—Either in its pure state, or in combination with zinc, it is of great benefit in cases of long standing, especially if there be debility and tardy restoration of power in the paralyzed limb. The phosphide of zinc in doses of one-third of a grain, or dilute phosphoric acid in half-teaspoonful doses, are perhaps better borne than pure phosphorus.

*Strychnine* is entitled to more consideration. If used at the proper time, it is more powerful to do good than any other remedy I know of, perhaps excepting electricity. When the exaggerated electro-muscular irritability subsides, we may give it in doses of 1-32 of a grain three times a day, but before this time its use is attended with danger.

Vance<sup>1</sup> has recommended hypodermic injection of strychnine, but I always hesitate when injecting an irritating substance into the belly of a paralyzed muscle, for I have repeatedly seen abscesses follow the use of even a neutral solution properly injected. Impaired muscular vitality and tardy reparative nutrition do not favor its use. However, Bartholow, Eulenberg, and Echeverria recommend its employment, and have had good results. Perhaps in paralysis of central origin the trouble to which I have alluded is not so much to be feared as when the affection is peripheral. Each muscle is to be subjected to injection, one being so treated each day. Instead of the plan recommended by these authorities, viz., injections into the substance of the muscle, I prefer local *subcutaneous* introduction of the solution by the hypodermic syringe. In addition to electric treatment, it is well to resort to massage and passive movement of the contracted members. The patient may be directed to do this himself, and he should be told to rub the paralyzed limb several times daily for at least fifteen minutes at a time. Dr. G. M. Beard has recommended heat in the treatment of paralysis, and his plan is to place the affected limb in a heated earthen drain pipe, well lined with flannel. I can quite agree with him, but have found that alternate heat and cold applied to the surface produce more rapid improvement in nutrition of parts which have lost their power. I originally recommended the instrument depicted in Fig. 22, which will be found a cleanly and convenient apparatus. One receptacle is filled with hot water, the other with cold. If the contracted limbs

Fig. 22.



Instrument for applying Heat and Cold.

where lately rigidity has taken place are allowed to remain daily for fifteen minutes or half an hour in quite hot water, much benefit will follow; or, should there be neuritis, we may use blisters, or the actual cautery along the course of the nerve trunk. It is of the utmost importance that everything should be done to improve the patient's hygienic surroundings, diet, and habits. He should not remain in-doors, but stay in the open air as much as possible. Food of a nutritious but not of a fatty character, moderate stimulation if needed, and a course of tonics, may constitute our form of treatment during this late stage of the disease.

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<sup>1</sup> Journal of Psychological Medicine, April, 1870.



## CHAPTER III.

## DISEASES OF THE CEREBRUM AND CEREBELLUM (CONTINUED.)

## SYMPTOMATIC CEREBRAL ANÆMIA.

**Synonyms.**—Syncope, Anémie Cérébrale, Hydrocephaloid.

**Definition.**—A morbid state characterized by an insufficient cerebral blood-supply, and expressed by impairment of consciousness, pallor, and much muscular enfeeblement. This disease is capable of quite as great modification as cerebral hyperæmia, as it may be what only appears to be a continued physiological condition, or a grave pathological state. Cerebral anæmia may occur: 1, in an acute form (syncope); 2, in a chronic form; 3, in an infantile form (the hydrocephaloid of Marshall Hall); and, 4, it is localized or partial, as a result of vascular obstruction. The acute form, which may be only a simple fainting attack, or the result of shock following severe hemorrhage, is the most familiar variety. It is hardly necessary to describe the alarming and familiar condition that we occasionally meet with after post-partum hemorrhage, or protracted decubitus, when the patient assumes the erect posture. The *chronic* variety is much less serious in its earlier stages, though, when continued, it is often the forerunner of certain forms of insanity. It is symptomatized by lowered function of the cerebral ganglia, depraved nervous tone, and general intellectual apathy; for, as normal circulation is necessary for the support of healthy brain action, and as we find that rapidity of thought and emotional activity are proportionate to the increase in the cerebral blood-supply, so must insufficient circulation bring with it an impaired state of intellectual functional activity. This loss of healthy action may be expressed by drowsiness, obscured intelligence, or by irritability and restlessness.

The *infantile* form generally follows some of the continued fevers of early life, and is a disease of childhood. Occurring during the stage of convalescence of the acute form, it is symptomatized by semi consciousness, diarrhœa, great exhaustion, insensitive pupils, pallor, sighing respiration, and other symptoms.

The last variety, *local* or *partial* cerebral anæmia, is that which is usually productive of right hemiplegia, and is due, in the majority of cases, to thrombosis or embolism, and often has a grave termination.

It is hardly necessary to allude to acute cerebral anæmia, for it comes within the province of the surgeon rather than within that of the neurologist. Following some grave accident when there is sudden and excessive loss of blood, we shall find a corresponding loss of consciousness, and muscular power, sighing, and slow respiration, generally vomiting, and involuntary discharge of feces and urine.

The condition is not a lasting one, and provided the hemorrhage has not been too excessive, nor the shock too great, there may be a retrograde disappearance of the symptoms, and ultimate recovery.

**Symptoms.**—A. CHRONIC CEREBRAL ANÆMIA.\*—Pallor of the skin, particularly of the face, which is of a dirty white color, while the sclerotics are milky blue, and the pupils widely dilated. The patient's expression is one of anxiety and depression, and if the condition be advanced and of long standing, he will spend hours with downcast eyes and a painful hopelessness, and hebetude stamped upon every feature. Coldness of the hands, heart-murmurs, and a weak, small pulse, are strong evidences of defective circulation of this description. The sphygmograph gives an almost straight tracing, the pulse-beats being weak and small. If the condition has gone on to the state where mental impairment has begun, we will generally find that there is venous stasis, and that the back of the hands is of a livid color, while pressure leaves a white mark which slowly disappears. The lips are pale, thick, and puffed, and the line between the mucous membrane and skin is less sharply defined than in the normal state. The urine is passed in large quantities, is colorless and limpid, and of a low specific gravity. The heart-sounds are weak, and it is not uncommon to find an aortic bellows murmur. Our patient complains of muscular debility, backache, loss of appetite, and somnolence, with great despondency, increasing loss of memory, marked headache, a regularly distributed cutaneous anæsthesia, sometimes nausea, hallucinations of sight and hearing, palpitation, indigestion, and constipation. I have been told very often by these patients that it was with very great difficulty that they could refrain from falling asleep in public places, and one lady was in the habit of becoming so drowsy in the street car on her way to my office that she very often unconsciously passed the street. Women who suffer in this way are subject to fainting attacks, which occur most often during the menstrual period. Among the most aggravating symptoms are hallucinations of hearing; noises—such as ringing of bells—are heard; and they occasionally have visual hallucinations in connection therewith. Delusions are very unusual. Insomnia is sometimes a distressing symptom, though during the day, as I have before said, the patient may have great difficulty in keeping awake. It is not uncommon for him to complain of a sensation as of falling through the bed; and one of the prominent elements of his sleeplessness is the continuous roaring in his ears, which is sometimes compared to the sounds heard when a shell or other hollow body is placed over the ear. There may be amaurosis, and other defects of vision. Digestive derangements are quite common, and vomiting, which is cerebral, is in some cases frequent and obstinate. The individuals presenting these symptoms are poorly nourished. There may be œdema of the legs and ankles, and sometimes albuminuria.

\* This term is used with caution, as it will not do to be too positive in making a diagnosis unless we are sure of the existence of some general cause. There are undoubtedly many cases of chronic cerebral anæmia due to the existence of organic cerebral disease which present symptoms mistaken very often for those of functional disease.

Feebleness and want of muscular power, of a light grade, are often expressed; and the comfort of a sofa or easy chair is sought by the patient, who seems disinclined to take any exertion whatever.

B. INFANTILE CEREBRAL ANÆMIA.—Marshall Hall has called attention to a most interesting form of anæmia, to which I have casually referred, and to which he has given the name "Hydrocephaloid." The disease depends principally upon exudation, and has its origin in early infancy. A case is related by Hall:—

"The patient, a boy, aged four, became comatose and perfectly blind and deaf. The finger might approach the half-closed eye without inducing any movement, but the moment it touched the eyelash, the eyelids would close. A spoon applied to the lips excited their action, and the food it contained was carried into the pharynx and swallowed; the respiration was frequently suspended; a sigh, and frequent respiration followed. The cerebral functions had ceased; the true spinal functions were made."<sup>1</sup>

Marshall Hall lays down certain rules from which I may extract the following. We should especially be upon our guard not to mistake the stupor or coma into which the state of irritability is apt to subside, for natural sleep, and for an indication of returning health. "The pallor and coldness of the cheeks, the half-closed eyelid, and the irregular breathing, will sufficiently distinguish the two cases." He divides the affection into two stages, the first of which is one of *irritability*, the second, of *coma*. In the former there is some attempt at reaction, and in both stages there is some resemblance to acute hydrocephalus.

"In the first stage the infant becomes irritable, restless, and feverish; the face is flushed, the surface hot, and the pulse frequent; there is an undue sensitiveness of the nerves of feeling, and the little patient starts on being touched, or from any sudden noise; there is sighing, and moaning during sleep, and screaming; the bowels are flatulent and loose, and the evacuations are mucous and disordered. If through an erroneous notion of this affection nourishment and cordials be not given, or if the diarrhoea continue either spontaneously or from the administration of medicine, the exhaustion which ensues is very apt to lead to a very different train of symptoms. The countenance becomes pale, the cheeks cool or cold; the eyelids are half closed, the eyes are unfixed and unattracted by any object placed before them; the pupils are unmoved on the approach of light; the breathing, from being quick, becomes irregular, and affected by sighs; the voice becomes husky, and there is sometimes a husky teasing cough; and evidently, if the strength of the little patient continues to decline, there is crepitus or rattling in the breathing; the evacuations are usually green; the feet are apt to be cold."

It is my opinion that this form of disease is very much more common than it is supposed to be, and that many deaths usually reported as *marasmus* are evidently of this nature.

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<sup>1</sup> Op. cit., p. 181.



Of *local cerebral anæmia* I will speak in another chapter.

**Causes.**—As causes of cerebral anæmia we may roughly class all agents that interfere with the cerebral blood-supply, and consider them as *remote* or *local*. Whether the fault lies in a diseased heart, which is unable to supply the brain with its normal amount of blood, or whether there is some mechanical obstruction through pressure upon the cerebral arteries, the morbid condition is the same. By far the most common cause of this cerebral condition is a general anæmia which may be dependent upon a number of conditions which drain the vessels. Among these may be enumerated uterine hemorrhages of various kinds, hemorrhoidal fluxes, cancers and other diseases attended by hemorrhage, as well as general diseases of assimilation which prevent the proper enrichment of the blood. A very slight reduction in the quantity of the blood will be followed usually by indications of the want felt by regions deprived of their nourishment; but when the nervous system suffers this deprivation, the loss is *immediately* shown. Haller has calculated that one-fifth of all the blood in the body is sent to the brain, and with this fact in view, it will not be difficult to realize how any modification of circulation will result in immediate changes. Heart disease generally in the form of fatty enlargement when there is mitral stenosis, or when functional activity is interfered with by emotional or other causes, may have much to do with cerebral anæmia. This cause enters, perhaps, more extensively into the production of chronic cerebral anæmia than any other. Owing to the delicate arrangement of the vaso-motor nerves which so beautifully control the supply of cerebral blood, when through emotional or other causes the function is altered, there will be immediate intra as well as extra-cranial anæmia. We have all seen that sudden emotions not only blanch the face, but as well produce faintness. Various changes in the functions of the liver may be associated with states of cerebral anæmia through modification of function of this system of nerves. Milner Fothergill has pointed out the association between the nerves of this organ and those which supply the vertebral arteries; and Schröder Van der Kolk and Laycock have held that those parts of the brain supplied by the vertebral arteries were the seat of the emotions. Fothergill reminds us of the fact that we may have functional derangement of the liver without affection of the intellect, but with depressed emotional states. There are other forms of abdominal trouble, such as an overloaded rectum and uterine derangement, which coexist with melancholia and depression of spirits, and every practitioner has seen the wonderful elation of spirits which follows a free movement of the bowels after continued torpidity of the liver. The extension of the cerebral vaso-motor, and the involvement of other areas of blood-supply may, of course, make the condition a more extensive one, and disturbances of motility and intellection naturally ensue.

Pressure made upon the carotid or vertebral arteries by various tumors or growths, or sometimes by aneurisms, is a mechanical cause of cerebral anæmia of decided importance. I assisted at an operation several years ago where the carotid on one side was tied by Drs. Sands and Parker, of

this city. In less than twenty-four hours the patient died from extensive anæmia, owing to the failure of compensatory supply. Embolism is perhaps the simplest example of a cause of this kind. A detached vegetation or clot is washed into the circulation, up through the left carotid and into the middle cerebral artery for instance, cutting off the circulation, and producing extensive cerebral anæmia on the left side, while right hemiplegia and aphasia follow. In thrombosis the artery is narrowed by the gradual deposit of plastic substances until finally its calibre is occluded, and the blood must take some other channel or not reach the part which it normally supplied.

Apoplexy, or brain tumors of various kinds, and atheromatous narrowing of cerebral arteries, are also direct causes. In the first two instances pressure is made directly on the brain substance, and in the latter there is a gradual change in the vessels themselves.

As a familiar illustration of how cerebral anæmia may be produced by a drain upon the general vascular system, I may allude to the case of a patient whose trouble dated from a series of miscarriages occurring within a very short period. One of these happened when it was impossible to procure medical attendance, and she lost a great quantity of blood.

After the last event she never completely recovered, and her present disagreeable and annoying condition remained. She was drowsy, had frontal headache, ringing in the ears; was constipated, etc. Another patient was subject to attacks of despondency, when life seemed very distasteful and gloomy. Her appearance was characteristic. White skin, cold hands, palpitation, and other symptoms enabled me to diagnose cerebral anæmia, and vomiting and vertigo were confirmatory symptoms. The cause was found to arise from very troublesome hemorrhoids. After cauterization and removal, she regained her previous health.

Certain medicinal agents, as well as tobacco, produce cerebral anæmia. The bromides undoubtedly possess this property, while chloral and chloroform, if taken for a long time, as they often are, are likely to provoke an anæmic state of the brain which is distressing in the extreme. I can recall the case of a young lady who confessed that she had been in the habit of putting herself to sleep at night with chloroform, besides inhaling it several times during the day. I have never seen such a typical case of this morbid condition. Her skin was of a hue of waxy whiteness, her pulse small and fluttering, her pupils widely dilated, and her languor and muscular feebleness very profound. Depression and the contemplation of suicide prompted her to confess her bad habit. Tobacco, though only affecting the heart, through its interference with pulmonary functions, undoubtedly produces in some individuals a condition of cerebral anæmia. The clammy, white skin, giddiness, dilated pupils, hurried respiration, and unsteady, weak pulse, and not uncommonly syncope, attendant upon nicotine poisoning, are, I think, evidences of cerebral anæmia. Certainly the after effects are clearly suggestive of this morbid cerebral condition. That tobacco, in many individuals, in fact the great proportion, possesses stimulating effects, there can be no doubt; but the

variation of effects which follows the administration of opium, for example, when there is some idiosyncrasy, clearly leads us to infer that its action is sometimes different from that determined by the majority of physiologists. Physostigma, veratrum, aconite, and like cardiac sedatives may be mentioned as other anæmiants.

Various conditions, such as lithiasis, are sometimes unsuspected, but nevertheless very important causes of cerebral anæmia.

**Morbid Anatomy and Pathology.**—As we might expect, the anæmic brain is white, firm, reduced in bulk, and greatly changed. The vessels are empty, and there are no puncta visible when a cut is made through the white matter. We may find a distension of the perivascular spaces, the ventricles, and arachnoid spaces by fluids, and occasionally some thickening of the neuroglia.

I have spoken in another chapter of the circumstances which modify the cerebral circulation. It only remains for me to refer to the experiments of Kussmaul and Tenner, Burrowes, and others, who have devoted a great deal of attention to the experimental study of this subject. The experiments of the first two observers were made upon six adults and a number of rabbits. When the carotids of the human subject were compressed, pallor, loss of consciousness, slow respiration, and dilated pupils were produced, which disappeared when the pressure was remitted, and could again be produced at will. Tying of the carotids was followed by convulsions, unconsciousness, and death, when *post-mortem* examination revealed evidences of softening.

In the first experiments, when pressure was remitted, there were evidences of a secondary cerebral hyperæmia with flushing of the face. Obstruction of the artery on one side may produce loss of motor power on the other, with immediate giddiness, loss of consciousness, syncope, and occasionally vomiting. There *may* be complete recovery after such an accident, but "it is always imperfect when the obstruction is situated on the further side (from the heart) of the circle of Willis."<sup>1</sup> The obstruction of the minor cerebral arteries, is followed by less complete intellectual derangement, by more marked vomiting and giddiness. Should the anæmia be quickly produced, as it is when severe injuries have been received and the patient literally "bleeds to death," convulsions form a prominent and almost constant symptom. Sighing respiration, and the other phenomena I have already named, are also expressed.

In cerebral anæmia there is impairment of functional activity, while in congestion the reverse is the rule. *Post-mortem* examination shows that the brain in cerebral anæmia is white, condensed, and less bulky, and the vessels are empty.

We have already cited the causes of cerebral anæmia, and it now remains for us to consider the part they play. Cerebral anæmia depends upon—

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<sup>1</sup> H. Jones, *Functional Nervous Disorders*, p. 66.



1. The insufficiency of cerebral blood-supply, through actual deficiency.

2. The action of certain agents upon the nerve-filaments themselves.

It is hardly necessary to again more than allude to the first of these. In this condition the effect of posture is said to greatly influence the cerebral state. The erect position is conducive to an aggravation of the symptoms, while recumbency favors the flow of blood to the brain. This relief follows the supine position when the individual has an ordinary attack of syncope. Abercrombie relates a case which is quoted by Fothergill, and which is, I think, a beautiful practical example of this change. The patient, who was greatly reduced by some gastric disease, gradually became deaf, but heard perfectly well when he lay down or stooped forward. As soon as his face became flushed, the improvement in hearing began, and when he raised his head the blush faded away, and he relapsed into his old condition. Abdominal paracentesis is followed by syncope, if the patient is not made to assume the supine position, for during ascites the abdominal veins are so impinged upon that when pressure is remitted they are capable of suddenly receiving a very large quantity of blood—in fact, so much as to deprive the brain, and produce anæmia. A quantity of blood gravitates directly through the superior and inferior venæ cavæ, not being thrown over by the right ventricle, but passing down into the abdominal vessels.

Insufficiency of cerebral blood may be due to a powerless heart, or aortic insufficiency, that organ being unable to lift a requisite amount of blood for the nutrition of the brain. Not only may this be a direct result of a weakened organ, but it may follow strong emotional excitement.

This assumption of the recumbent posture is one of the best therapeutical means in certain cases. Dr. Weir Mitchell has had extraordinary success in the management of certain intractable cases, some of which were directly dependent upon cerebral anæmia.

Of the second mode of production, I may allude to the local effect of some blood poisons, and the influence of the emotions. Bearing in mind the important physiological law that section of the sympathetic is followed by vascular dilatation, and that irritation of the proximal end produces contraction, we are enabled to realize many of the pathological processes which occur in the production of cerebral anæmia. Anteriorly the vasomotor fibres are derived from the superior cervical ganglion, and posteriorly the fibres come from the inferior cervical ganglion. These filaments follow the course of the large cerebral vessels, and in this manner supply every part of the cerebral mass.

This close relation with the vascular system explains the prompt action upon the heart of certain exciting emotions, and secondarily the variation in blood-supply. This is the idea held by Fothergill and others, and most admirably explained by that writer in an article in the *West Riding Reports*.<sup>1</sup>

<sup>1</sup> Art. Cereb. Anæmia, vol. iv., p. 108.

The connection between variation in cell action and the function of the sympathetic fibres is, perhaps, the most interesting part of the subject. Primarily the influence of impoverished blood affects the integrity of the cerebral nerve-cells, and secondarily the influence of the cerebro-spinal fibres is suspended. I have no doubt that a certain train of symptoms, which is sometimes expressed during general anæmia, is the result of a temporary local hyperæmia, through paresis of the vaso motor fibres; and that parts of the brain are congested while others are anæmic.

A result of continued emptiness of the vessels is an œdematous condition of the brain, from distension of the perivascular spaces by the cerebro-spinal fluid. This condition is sometimes so extensive as to receive the name "serous apoplexy," and profound stupor is the result.

In relation to sleep and its connection with cerebral anæmia, it will bewell to say a few words. A great many observers, among whom were Durham and Fleming, strongly held that the brain is anæmic during repose, the anæmia being the cause of sleep. Others have differed with them; and experimental facts seem to favor this view of the case. Not only may anæmia be unattended by sleep, but a condition of unconsciousness closely resembling healthy sleep may be the result of a hyperæmic cerebral state. Opium, alcohol, and various agents which increase the cerebral blood-supply, act in this way; but the stupor which follows a toxic dose of either agent must not be confounded with natural sleep. Certain curious facts militate strongly against the anæmic idea, or, at least, against the assertion that sleep is directly dependent upon a diminution in the supply of blood to the brain.

1. There are many anæmic individuals who sleep only after taking stimulants. I think all who have seen the good effects of a bottle of ale at bedtime will be disposed to take this view. The sleep produced in no way resembles stupor, and there is no disagreeable sense of fatigue in the morning.

2. Dr. Janeway made an interesting experiment. This consisted in the administration of a few drops of nitrite of amyl to a sleeping person. Although cerebral congestion followed, the patient did not awake.

3. If mental action is dependent upon activity of the cerebral circulation, and sleep upon anæmia, it almost seems that dreams must be inconsistent with sleep; while, on the contrary, many individuals enjoy the most vivid and constant dreams, and do not awake till their usual hour.

I am more inclined to think that the production of sleep depends upon some change in the function of the nerve-cell, and that this modified form of action is not necessarily dependent upon either anæmia or congestion in any particular case, *but that, if there be anæmia, it is secondary to the cell-change, whatever that may be.*

The connection of a torpid condition of the liver with cerebral anæmia will explain the constipation, which is anything but an uncommon accompaniment of the disease. Intestinal accumulation, as Fothergill says, may "stand to cerebral anæmia in a causal as well as a consequential relationship," and he alludes to the experiments of Ludwig and Daziel to

illustrate the connection. A finger passed over the intestines produced acceleration of the intracranial circulation.

The general symptoms, such as languor, the various modifications of sensation, etc., are directly due to a diminution in nervous supply.

**Diagnosis.**—Acute general attacks of cerebral anæmia may be confounded with cerebral congestion, stomachic and auditory vertigo. I have already spoken of the distinction to be made between the disease under discussion and cerebral hyperæmia, and it is not necessary to say more. Attacks of stomachic vertigo, or Menière's disease, are symptomatized as follows: The first is characterized by a feeling of "emptiness of the head," reeling and swimming, general coldness; "objects whirl around;" *no loss of consciousness*, nor marked disposition to sleep. No dependence upon a very full or empty stomach, and the possible existence of gastralgia. In Menière's disease there is aural disease, and turning or whirling generally to one side, from left to right, and the condition is not continuous. The most important facts to discover are in relation to the cause, whether it be a secondary condition, the result of cardiac trouble, or whether it be simply a result of general anæmia, without any organic disease.

Chronic cerebral anæmia presents various phases, and it is almost impossible to go over the long list of general diseases which it may be a feature of, or, which, like hysteria, it may counterfeit. Cerebral tumor may give rise to symptoms which are really due to cerebral anæmia. So perfect is the resemblance that Dr. Hughlings Jackson told me recently that it would be impossible for him to make a diagnosis in many cases with any degree of certainty.

**Prognosis.**—As cerebral anæmia is nearly always due to some cause which is easy of removal, the prognosis is good. If, however, there be organic heart trouble, the case assumes a different aspect. Old cases are extremely discouraging, particularly when the patients happen to be women. Irritability and hysteria generally enter largely into the complaint, and treatment is sometimes almost useless. If uterine, hemorrhoidal fluxes, and other such drains, exist, of course their amelioration is attended by cure. Should the loss of blood be caused by a cancerous uterus or rectum, the prognosis is consequently very bad.

**Treatment.**—It is of the utmost importance that the practitioner should seek out and remove, if possible, such conditions as diminish the amount of blood in the body, and consequently he must ascertain the existence of hemorrhoids, uterine hemorrhages, either periodical or irregular, and apply appropriate remedies in such cases. Without venturing upon another field, I would call attention to the necessity, in cases where there is menorrhagia, of overcoming this condition as promptly as possible, for special treatment of the nervous condition is of little avail when the woman every month loses a quantity of blood largely in excess of what is made in the interim.

I have, of late, had encouraging success in the treatment of cerebral anæmia by means of nitrous oxide gas.

This gas is essentially a nervous stimulant, and while its action is



somewhat like that of oxygen, it has the advantage of influencing the intellectual and emotional functions.

The use, say of two gallons of gas mixed with one of air, will produce pulse quickening after two or three full inhalations, and such quickening will be attended by very slight flushing of the face, and throbbing of the temporal vessels.

If the administration be carried sufficiently far a condition of temporary unconsciousness results, which is attended by anæsthesia, and upon recovery, there is a certain amount of reaction. It is unnecessary to say that the extension of the effects of the gas to this stage is entirely out of the question, and an extremely injudicious measure when the desire is to improve circulation and nutrition.

Exhilaration of spirits is the rule after its use, not however, necessarily amounting to the *abandon* that so often follows the lecture room experiments of ten or fifteen years ago, but sufficient to indicate a very decided activity of ideation and the emotions. Melancholic and taciturn subjects became animated and cheerful in their address and behavior. One of the patients, of the late Dr. J. Ellis Blake who first used the gas in America as a therapeutical agent in nervous disease, declared that the figures upon his ledger bore an entirely different import after he had taken his dose of gas, and walked to his office, and the debit side looked wonderfully less depressing. In another case, the patient who had left home quite reluctantly, and desired at first to go back immediately, forgot all his worriments after the first two or three days of treatment. It is certain that in hypochondriacal patients many minor aches and pains are forgotten, and a general *couleur de rose* tinges everything.

My attention was forcibly drawn to this effect upon certain patients after I had used it with melancholics, both in my private practice and at the Insane Asylum at Blackwell's Island. One of these had suffered for several weeks from the most profound despondency. Her trouble had grown out of menstrual irregularity, and was evinced by religious delusions of a mild type, inclination to avoid the society of her friends, and an occasional refusal to eat. The use of the gas for several weeks entirely removed her mental trouble, and she became quite cheerful. In the presence of Drs. MacDonald, Pitkin, and Lesynsky, nitrous oxide was given to two melancholic patients at the Female Insane Asylum who had refused food, and had not eaten voluntarily for two weeks. Both of the women went to the table and ate heartily the same evening.

In other cases of melancholia with defective surface circulation, the venous stasis which gave the hand a dusky purple color disappeared in a few days to a great extent, and the white mark which remained after pressure of the finger upon the back of the hand had been remitted, did not last nearly so long, nor was it so sharply defined as under other circumstances. The warmth of the extremities was decidedly increased, and the expression of the eyes was brighter, and much more intelligent.

Mitchell <sup>1</sup>reports seven cases of melancholia, mania and dementia treated with nitrous oxide, in all of whom interesting effects were witnessed. The gas was not administered however, for its stimulant effects alone, but given until the point of partial unconsciousness was reached.

Active measures are necessary when there is general anæmia, and for this purpose we must resort to iron, strychnia, phosphorus in some of its forms, cod-liver oil, an abundance of nutritious food, with stimulants such as milk punches, porter, or ale.

A word or two is necessary in regard to the diet, and the quantity of alcohol given to these patients. It is the physician's bad fortune to meet with cases of this kind in which digestive troubles are dependent entirely upon an enfeebled state of the viscera, and we should therefore use great care and not be impatient. A hearty regimen, and too much alcohol, may do mischief instead of good. It is well, therefore, in certain cases, to give the stomach as little work as possible, and at the same time to allow it to exert itself in a way that will most benefit its possessor. A very little food, given at short intervals, will be more perfectly digested and assimilated than a large quantity taken at long intervals. I have often given a few table-spoonfuls of cream or beef-juice every hour for days, and have ultimately seen such a marked improvement and an increased capacity for work upon the part of the digestive organs, that the more gross varieties of animal food, as well as alcohol, were after a while borne in large quantities. Should this enfeeblement of the digestive organs exist, we may give either pancreatine emulsion, or strychnia and muriatic acid. Extract of malt is sometimes very well borne, and hastens the improvement. This may be given in combination with codliver oil.

One of the most useful forms of treatment to which I have already alluded—the “rest treatment” of Weir Mitchell—is of marked service in old cases, especially if the subjects happen to be women. Dr. Mitchell has treated many cases which are almost identical with those that generally come under the head of chronic cerebral anæmia. He says: “These cases vary, of course, endlessly; but their essence is a state of reduced nutrition, which no mere tonic will cure, while they are afoot and living on their capital. The main symptoms are the state of painful tire, the low temperature, the great or less anæmia, the quick pulse, the excess of white blood.” He calls attention to the necessity for perfect quiet, and at the same time daily massage and faradization of all the muscles. His treatment is expressed in his own words thus: “The amount of feeding, of massage, and of faradic-muscle exercise which each case will bear and prosper under, is a matter to be told early in the case by watching the pulse, the temperature, and the appetite. In these cases the pulse is always rapid. If it fall, if the temperature rise, above all, if there be the least gain in flesh, I know that I am on the right path and am not moving on it too fast; but if these symptoms be reversed, and if the patient ceases to be hopeful and looks weary, then I lessen the passive exercise, and wait a little; but, above all, I listen to what my

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<sup>1</sup> W. R. Reports.

masseur or masseuse tells me of the ease with which the limbs flush or the readiness with which the muscles grow firm under the kneading fingers, for in this matter I get to have a very shrewd judgment. As to the rectal feeding, which I rarely omit, I say little, as it is well understood. It should always include cod-liver oil. There is only this to be borne in mind: most medical men feed by the bowel when they cannot by the mouth. I like to use both ends at once."

This treatment seems to be the very best in cases of long standing; but it is well to see first what fresh air, tonics, and abundant nitrogenous food will do for our patient, while she pursues her ordinary life.

The selection of a climate for the nervous patient is a matter of great importance. Dr. Denison, of Denver, who has written much upon this subject, and who has lived in Colorado, speaks with some caution regarding the benefits of high altitude. He says: "The more acute or severe the nervous symptoms, the more of an aggravating nature is the effect of an elevation."<sup>1</sup> He does not recommend Colorado for patients who suffer from epilepsy or cholera, but only in such cases where the diseases of the nervous system depends upon certain dyscrasia. Organic diseases are aggravated. In cases of nervous exhaustion with anæmia and depression, there can be no doubt of the advantage of the stimulating climate of Colorado, and to such a place we might send our patients, expecting great benefit.

### STOMACHIC VERTIGO.

**Synonyms.**—Vertigo a stomacho læso (Lat.); Vertige stomacal (Fr.); Gastric vertigo.

**Definition.**—A condition of giddiness, hallucination, nausea, headache, etc., without loss of consciousness, and probably dependent upon a reflex excitation of the cerebral vessels from some visceral irritation.

**Symptoms.**—The condition, which is a very common one, is produced, in most cases, directly after a hearty meal, or else when the stomach is entirely empty. A sense of gastric fulness at first, while headache, with buzzing in the ears, palpitation, and giddiness of a few moments' duration, follow. Should there be hallucinations, the patient is not worried by them, but realizes their unsubstantial character. Trousseau<sup>2</sup> insists upon the fact that the hallucinations of this condition differ from those attendant upon cerebral hyperæmia from the fact that in this form they do not occur when the head is *lowered*, which is the case in cerebral hyperæmia.

**Causation.**—Stomachic vertigo is more a condition of middle life and old age than one of youth. Young women occasionally suffer, but this is the exception. Certain forms of indigestible food may directly pro-

<sup>1</sup> Rocky Mountain Health Resorts, p. 145. .

<sup>2</sup> Clinical Medicine, Am. edition, vol. ii. p. 358.



voke the attack, or it may follow violent exercise after a hastily eaten meal. In one case of which I know, a gentleman ran for over a mile to catch a morning train. He had arisen but a few moments before, and had hurriedly eaten his breakfast. He fell to the ground, but did not lose consciousness. The disorder often occurs when the individual has been eating irregularly; and business men or others who take but little exercise and eat hurriedly are very often the sufferers. Handfield Jones<sup>1</sup> considers tænia to be a frequent cause of vertigo, and such has been my own experience.

**Treatment.**—Trousseau, who has written most fully upon the subject, recommends that the patient be directed to drink every morning a glassful of quassia infusion made by maceration of the shavings in water, or to use the goblet of quassia wood in which the water is allowed to remain until it has become bitter. After each meal one of these powders should be taken:—

R. Sodæ bicarb.,  
Magnesiæ calc., āā gr. xv.  
Cretæ præp. ʒss.—M.

Divid. in chart. no. iij.—Sig. One after each meal.

Strychnia, pepsine, and sometimes bismuth are excellent remedies, and should be given, while attention is to be paid to the patient's general habits.

## AUDITORY VERTIGO.

**Synonyms.**—Labyrinthine vertigo; Mènière's disease.

**Definition.**—A morbid cerebral condition expressed by vertigo and rotatory movements, unattended by loss of consciousness, and dependent upon disease of the labyrinth, or other parts of the central auditory apparatus

To Mènière<sup>2</sup> belongs the credit of having first accurately described this disease, though Triquet<sup>3</sup> gives the credit of its discovery to Saissy, of Lyons, who observed a nervous condition connected with diseases of the inner ear. Trousseau<sup>4</sup> says that Saissy did not mention vertigo as a symptom of the condition to which he called attention. It is enough to say that, prior to 1861, the form then known only as stomachic vertigo was always supposed to arise from digestive troubles, and the existence of a distinct variety, with aural disease, was not appreciated.

**Symptoms.**—Generally there are some indications of otitis, whether they be simple inflammation denoted by pain, or a discharge of bloody pus, or even perforation of the tympanum. In many cases the disease

<sup>1</sup> Functional Nervous Disorders, p. 444.

<sup>2</sup> Bulletin de l'Académie de Méd., xxvi. p. 241.

<sup>3</sup> Leçons cliniques sur les Maladies de l'Oreille, p. 113, Paris, 1863.

<sup>4</sup> Loc. cit., p. 363.

may be preceded by a chill, and this should be always looked upon as a serious indication. The patient is suddenly seized with vertigo, and at the same time experiences a feeling of nausea and buzzing in the ears, which may be double, or confined to one side. This vertiginous condition calls to mind a sensation experienced when one is twirled in a swing. A boyish prank is to twist the ropes of a swing while the unhappy victim is seated therein; then to suddenly release the board, which revolves with great rapidity as the ropes unwind. This description of the symptom was given me by a patient who suffered from nausea at the same time with vertigo. The vertigo is attended by a loss of equilibrium. The patient sways or reels, and there is an impulse to turn from the left to right when the left ear is affected, and *vice versa* when the other is the seat of the disease. Ferrier<sup>1</sup> describes a sensation usually experienced. He (the patient) feels "as if he were suddenly lifted from the ground and pitched forward and to the right side." There is also a tendency, when walking, to keep close to the side of the wall or house which corresponds to the affected ear. Deafness is generally present, but this is, of course, the result of the destructive aural disease.<sup>2</sup> Recovery is not always to be expected, but a great many cases improve under appropriate treatment presently to be described.

John B., aged 47, iron railing manufacturer. Nearly eighteen months ago, he became troubled by noises in the left ear, which he compared to the "singing of canary birds," and afterwards this subjective noise changed its character, and he described it as a continuous roaring like the escape of steam from a boiler. To this sound he has since become partially accustomed. He has never had earache, but nine years ago there

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<sup>1</sup> Labyrinthine Vertigo, W. R. Reports, vol. v. p. 34.

<sup>2</sup> Crum-Brown is of the opinion that, in addition to the other senses, the individual possesses one of rotation, by which we are able to determine the axis about which rotation of the head takes place; the direction of rotation, and its rate. In explaining some experiments performed by him, he says: "In ordinary circumstances we do not wholly depend upon this sense for such information. Sight, hearing, touch, and muscular sense assist us in determining the direction and amount of our motions of rotation, as well as of those of translation; but if we purposely deprive ourselves of such aid, we find that we can still determine with considerable accuracy the axis, the direction, and the rate of rotation. The experiments that I have made with the view of determining this point were conducted as follows: A stool was placed on the centre of a table capable of rotating smoothly about a vertical axis; upon this the experimenter sat, his eyes being closed and bandaged; an assistant then turned the table as smoothly as possible through an angle of the sense and extent of which the experimenter had not been informed. It was found that, with moderate speed, and when not more than one or two complete turns were made at once, the experimenter could form a tolerably accurate judgment of the angle through which he had been turned. By placing the head in various positions, it was possible to make the vertical axis coincide with any straight line in the head. It was found that the accuracy of the sense was not the same for each position of the axis in the head; and, further, that the minimum perceptible angular rate of rotation varied also with the position of the axis. It was also found that considerable differences of accuracy exist in different individuals."

was a discharge from the left ear, but there have since been no other symptoms. He has suffered for a long time from post-pharyngeal catarrh, and there is now a catarrh of both Eustachian tubes. When a young man he had secondary syphilitic symptoms, but denies having had any primary sore. Sixteen months ago, during hot weather, he was seized in the street with dizziness and reeling, and was obliged to grasp a lamp-post for support. There was no loss of consciousness, and he realized fully his condition of helplessness. He said that he felt as if he was being "twirled" from right to left, but did not fall. This attack occurred before dinner (about 11 A. M.), and his stomach was neither filled nor completely empty, for he had eaten his breakfast at 8 A. M. He was perfectly well otherwise, and the only disordered function was that of the lower bowels, for he was constipated. He has had these attacks very frequently. For the six months following the first attack of vertigo they occurred about once a month, but since then they had been of daily recurrence.

*Present State.*—The patient's digestive organs are in good condition, and his appetite is fair. He is ordinarily of constipated habit, but it requires but slight medication to overcome this. He is of medium height, weighs 143 pounds, and seems a well-nourished man. His face is somewhat suffused when he becomes excited, but he is ordinarily pale. His eyes convey an anxious expression, but the pupils are normal. His hair is scanty and gray, but not removed in patches, nor suggestive of any previous syphilitic trouble. He has occasional headache, and still complains of the "roaring" noise on the left side. Hears the tick of a watch only six inches from left ear, and indistinctly at any distance within this limit. Watch tick heard at five inches from right ear, but more perfectly. Dr. C. S. Bull examined his eyes, and the following is his report:—

"Examination of J. B.  $V = \frac{20}{40+}$ : with convex 32 spherical  $V = \frac{20}{40+}$

H  $\frac{1}{30}$ . Fundus perfectly normal."

His attacks occur nearly every day, and seem to have no relation with the condition of digestion. These "reeling fits" may take place at any time of the day, last for five or six minutes, and usually are not so sudden as to prevent him from taking hold of the nearest lamp-post or railing. In a recent vertiginous seizure he was taken just as he was about to get into a street car, and would have fallen had the conductor not dragged him upon the step. He tells me that he has asked his wife to "turn him the other way" when the attack occurs, and usually this has the effect of abating it. I placed him upon large doses of quinine at first, which have decidedly influenced the frequency and character of the vertigo, so that he often passes a week at a time without any seizure. Bromide of potassium had been prescribed for him before his visit by another physician, but he tells me that this drug increased the dizziness. The phenomena of these attacks are the following: He suddenly feels light headache; objects swim about him from right to left while he seems to be rotated the other way, and during this period he separates his feet and braces himself. The outlines of the houses, trees, and sidewalks are blurred and distorted, and after a few minutes they suddenly assume their proper relations, and the attack passes off, and he has subsequent headache.



**Causes.**—The disease being directly due to aural inflammation, and the causes of this condition, whether they be exposure, the extension of other inflammatory processes, or the injudicious use of douches and injection, are only secondarily productive of the neurosis.

**Pathology.**—The experiments of Flourens and Goltz<sup>1</sup> have been the basis for our pathological study of Ménière's disease. Brown-Séquard<sup>2</sup> and Flourens demonstrated that when the membranous canals of the labyrinth were divided, various disturbances of equilibrium followed. Walter and Lincke<sup>3</sup> and others have divided the horizontal canals and produced oscillation of the eyeballs, swaying of the head from one side to the other; and have seen the animal spin round like a top. Division of the posterior vertical canal causes the animal to topple over backwards, and the head is moved backwards and forwards. When the superior vertical canals were cut across, the animal pitched forward. It may be seen that a diseased condition, not limited to any particular spot, may produce a combination of these symptoms.

Brown Séquard, in speaking of the relation of rotary movements to auditory irritation, calls attention to these familiar illustrations:—

“1st. Any one who has received an injection of cold water in the ear may know that it produces a kind of *vertigo*, and that it is difficult to walk straight for some time after this irritation. 2d. A sudden noise makes the whole body jump, particularly in old people, or in persons attacked with anæmia, chlorosis, epilepsy, chorea, hysteria, hydrophobia, and in certain cases of poisoning; in a word, in all circumstances in which the control of the will over reflex actions is lost or diminished. 3d. Vertigo and various convulsive movements in cases of irritation of the acoustic nerve have been observed in adults and children. Rotatory movements have taken place in cases of suppurative inflammation of the ear, and twice immediately after an injection of nitrate of silver.” Ferrier,<sup>4</sup> who has written most clearly upon this disease, goes very deeply into the subject. In the normal state it is necessary for tactile, visual, and auditory impressions to be unembarrassed, so that the power of equilibration may be preserved; but it is of absolute importance that the labyrinthine functions should be perfect. It seems to regulate the state of equilibrium of the individual, and to preside over co-ordination. The mechanism of the labyrinthine canals is admirably described by Crum-Brown.<sup>5</sup> The sense of rotation, as suggested by him, must, like other special senses, have a special peripheral organ, a brain centre, and a connecting sensory nerve. All experimenters agree that the labyrinth is a special peripheral organ, and the auditory nerve is that which conveys the peripheral irritation to the centre.

<sup>1</sup> Pflüger's Archiv für Physiologie, 1870, and Recherches sur les Propr. et les Fonctions du Système Nerveux 2d. ed.

<sup>2</sup> Central Nervous System, Philadelphia, 1860, and Experimental Researches, 1853.

<sup>3</sup> Wagner's Handwörterbuch der Physiol., vol. vi., 1853, p. 420 et seq.

<sup>4</sup> Ferrier on the Functions of the Brain, New York, 1876.

<sup>5</sup> Journal of Anatomy and Phys., May, 1874.

"The bony canals are filled with liquid, in which float loose connective tissue, and the membranous canals with the contained endolymph. Rotation of the head about an axis at right angles to the plane of a canal will then produce, on account of the inertia of the liquid, etc., motion of the contents relatively to the walls of the canal; and this may be expected to irritate the terminations of the nerves in the ampulla. If the rotation be continued at a uniform rate, fluid friction of the endolymph against the membranous canal, and of the perilymph against the membranous canal, and the periosteum will gradually diminish this relative motion, which will at last cease. We should therefore expect, as we have seen to be the case, that continued uniform rotation should be perceived less and less strongly, and that the sensation should at last die away altogether. The time required for this equalization of the motion of the canal and its contents will depend upon the rate of rotation and upon the dimensions of the canal and the amount of attachment of the membranous canal to the periosteum. These latter conditions are not the same in the three canals, and therefore we ought to find, as we do, that the rate at which the sense of rotation dies away is not the same for different positions of the head. Again, if the uniform rotation is stopped, the contents of the canal will continue to move on, thus causing an apparent rotation in a direction the reverse of that of the original rotation, and this also will die away owing to friction." The irritation of the auditory nerves which occurs is attended by anæmia of certain parts of the brain, which accounts for the reeling, dizziness, nausea, and other symptoms with which we are already familiar.

**Diagnosis.**—Gowers,<sup>1</sup> in a paper before the British Medical Association, pointed out the liability of its confusion with gastric trouble. He calls attention to the fact that violent and repeated vertiginous attacks, the sense of movement or actual turning, tinnitus aurium, and deafness, are more suggestive of the auditory origin than of gastric vertigo. Gowers' cases were connected with affections of smell and taste, and at the same time in one there was a gastric ulcer. He made his diagnosis by the detection of loss of function of the right ear and by one-sided falling. It is often necessary to differentiate from *petit mal*, from apoplectic warnings, and from general cerebral anæmia. In the first there is rarely vertigo, but there is loss of consciousness of temporary duration, and there is some convulsive movement, though sometimes so slight as to be unrecognized. The presence of aural disease is enough to throw out of the question the other condition I have named.

**Treatment.**—Large doses of quinine have been of service in these cases, and Charcot's<sup>2</sup> experience with this agent is extremely gratifying.

He recommends the energetic use of revulsives in vertigo, the cautery being applied over the mastoid bone three or four times a week. He gave sixty centigramme doses of quinine in one case for a period of two months with happy results, and a short time after the commencement the

<sup>1</sup> Br. Med. Journal, Aug. 26, 1876.

<sup>2</sup> Leçons sur les Maladies du Syst. Nerv. No. 4, p. 321.

vertiginous attacks ceased. It is necessary to give the drug in large doses, and at the same time the aural disease should not be neglected.

In the case of "J. B." I combined infusion of digitalis with the quinine, and obtained very good results. He was also directed to turn in an opposite direction to that caused by the disease. Subsequent experience has convinced me that strychnine is perhaps better than quinine, and I have been highly successful in relieving a case of much greater violence in which increasing doses of the drug were administered. In this connection it will be well to call attention to attacks of malarial vertigo of a periodic character which are sometimes encountered, and which resemble auditory vertigo: quinine or arsenic is of course indicated.



## CHAPTER IV.

## OCCLUSION OF INTRACRANIAL VESSELS.

## THROMBOSIS—EMBOLISM.

THE deprivation of an area of greater or less extent of its blood-supply constitutes a condition which has been called by some writers "Local cerebral anæmia," and it may take place through the existence of either of the above vascular states. Though very closely allied, these two forms of mechanical obstruction may be defined: in one case, as the *local* formation of deposits, or morbid changes favoring obliteration of blood-vessels; and in the other, as the lodgment of clots, or organized tissues which have been brought from a distance. Their chief interest lies in the fact, that it is often difficult for us to distinguish the subsequent symptoms from those indicating an effusion of blood from a ruptured vessel; that speech troubles are prominent; and that the prognosis is nearly always unfavorable. Thrombosis and embolism, though usually followed by many of the same symptoms, and confounded with each other by some of the medical writers by whom they were first described, differ greatly in their manner of occurrence and pathology. The first, as we shall hereafter see, is of slow development, and is not so serious in its results as embolism, while the latter condition is much more grave in *all* its features.

## INTRACRANIAL THROMBOSIS.

Any local vascular change from the normal state which favors the deposition of fibrine in an intracranial vessel, whether it be an artery, a vein, or sinus, produces the condition which is known as *thrombosis*. As a consequence, the calibre of the vessel is narrowed, and circulation of blood is impeded therein; clots form, and either from actual obstruction of direct supply or by pressure, a region of greater or less extent becomes anæmic. Though the arteries are more frequently the seat of such an alteration, the veins and large sinuses and the capillaries may be plugged up by clots which are of local origin. The condition, however, last mentioned is fortunately a very rare one, but when it is met with it is a most dangerous and alarming morbid state.

## THROMBOSIS OF THE CEREBRAL ARTERIES.

**Symptoms.**—It is a disease of slow development, and may affect several arteries simultaneously, or but one. For weeks, or even months before, distressing and important evidences appear, and the patient may present unmistakable expression of the cerebral change, such as headache, which is generally localized, confusion of ideas, and awkwardness of speech, these disturbances being, usually, varieties of aphasia. As the disease advances, this trouble becomes much more pronounced, and, in place of there being simply a difficulty in expressing a clearly originated idea, there may be a condition of *amnesia*. Clumsiness of speech, and want of delicacy in articulation are followed by an actual failure in remembering words. Memory is also defective in other things, and one patient begins to become stupid and listless. The next indication of this advance may be the appearance of paralysis, which is sometimes slight, or incomplete, only involving the muscles of the face or eyeballs, or there may be hemiplegia. Should the thrombus be seated in a large artery, or softening occur, a complete and lasting hemiplegia may be produced. There is rarely loss of consciousness at any time, and in very few of the cases that recover, is there anything at all like the paralysis following cerebral hemorrhage.

Recovery is generally to be looked for, provided the vessel be not an important one; and, though like its first cousin, embolism, it may be one of the causes of softening, such a termination is not always to be feared. Aphasia, which is insisted upon by most writers as a pathognomonic sign, is occasionally absent. In one case reported, though the left middle cerebral was affected, there was no aphasia at any time.<sup>1</sup>

The following case is one that came under my observation, and is of interest, because of the seat of the thrombus, and the interesting character of the morbid appearances:

L. C., aged 22 years, seamstress; admitted into hospital October 9, 1876. History from friend who accompanied her. The patient had been feeling unwell for about two months, having had pains in her head and back, loss of appetite, insomnia, and other troubles. About a week ago the friend went up to her room to assist her to dress for breakfast. When the patient stepped out of bed she fell upon the floor, and then first noticed that she was completely paralyzed on the right side. The friend knew nothing of the patient's antecedents. Her husband, who was seen subsequently, stated that he had left her because she drank; and that after the separation she went to New York and became a prostitute. Two years ago he saw her, and at that time she had marks of syphilis on her face, and her hair was falling out. She conversed with him intelligibly, but said she was suffering from "general debility." She had headache, pain in the back, etc., and was at this time leading a very irregular life; sitting up during the greater part of the night, and sleeping only a portion of the day. She went to Ward's Island for treatment. The

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<sup>1</sup> St. George's Hospital Reports, vol. i., 1866, vol. vi., p. 322.

following history was taken by Dr. Naylor, resident physician in hospital:—

*Oct. 10.* Complete hemiplegia of the right side, limbs lax, and muscles flabby; impossible to excite reflex movements by tickling; right pupil irregular, and smaller than the left; tongue drawn to left side when protruded, and when she laughs the right side of the face is drawn up. Control over the sphincters good; temperature  $101^{\circ}$ ; patient aphasic. When asked, "How long have you been sick?" replied, "Since Benny;" this answer was given to many questions asked. "What do you hold in your hand?" (it was a piece of bread.) "Tobacco." Seemed puzzled, but when reminded of its true nature she brightened up and appeared to realize her mistake.

*13th.* In about the same condition. Muscles of the right arm and leg do not respond to the currents. When asked how old she was, replied, "So and so." "What did you work at?" "So and so." "What street did you live in?" Appears puzzled. "Was it *sixteenth? seventeenth? eighteenth?*" "Yes." "How long has it been since you last saw your mother?" "You long so, John." Expression intelligent, and she seems to understand all that is said to her. Does not hear so well on left side, with right ear perfectly.

*17th.* Appeared to be suffering great pain. When asked to locate the pain, she did not attempt to do so. She has passed no urine since yesterday morning. Has a hard and swollen erythematous spot on the outside of each knee, and two similar enlargements on each leg below. There is a hardened red spot over the fourth cervical vertebra. All of these parts are painful to pressure.

*18th.* Right hand somewhat swollen. 6 P. M. Is drowsy this evening. Appears to suffer pain, and places left hand upon abdomen. One pint of straw-colored urine containing no abnormal constituents was drawn by the catheter.

*19th.* Still dull and drowsy. Said nothing to-day but "yes," "no," and "well;" passed her urine in bed; stupid and dull all day. Carotid on right side pulsates very distinctly.

*21st.* Somewhat brighter to-day; bowels regular.

*22d.* Relapse to stupid condition; passed urine in bed; became choked while eating some beef at dinner.

*25th.* Still absolute loss of power and sensation on right side, and continued drowsiness.

*26th.* Involuntary discharges of feces and urine.

*27th.* She brightens up after receiving nourishment, but cries and seems distressed.

*28th,* 2 P. M. Nurse called the house physician, seeing that she appeared to have stopped breathing. Her eyes were turned upwards and her lips blue, and her pulse was very weak and feeble. Ordered stimulants.

*Nov. 2d.* Feverish and restless; temperature  $101^{\circ}$ ; discharges from the bowels have stopped.

*6th.* Complains of pain in her thigh and legs; cries a great deal; refuses food, and appears to be very much run down.

*8th.* Right pupil approaching more nearly the size of the left; appetite still good; bowels regular. Cannot write her name with the left hand, but makes a disorderly scrawl. Asked her to repeat several words; pronounced "eggs" very distinctly; for "cross," she said "cork." 7 P. M. Quite feverish and restless; temperature  $102^{\circ}$ .



13th. Has still fever; temperature  $102^{\circ}$ . Ordered quinine and cold sponging. She cries, and appears very sensitive when moved.

14th. Slept well last night. 7 P. M. Temperature  $100^{\circ}$ . Several inguinal glands on the right side are somewhat enlarged and painful on pressure.

22d. Complains of great pain at the attachment of the adductors to femur.

The month of December was passed without anything occurring of special note. The patient grew much more feeble; there was no improvement in the paralysis, and she became reduced to a shadow. The temperature continued elevated, and she was restless and delirious at times. Of course the burden of her delirium consisted of two or three words, which were repeated over and over.

Jan. 8, 1877. Dr. Naylor was called to see the patient at 4 o'clock P. M. He then noticed some fibrillary contraction about the right angle of the mouth, with an occasional spasm of the upper lip, when it would be drawn up with the wing of the nostril. Eyes closed, pupils more contracted than usual, face flushed and head hot; temperature in axilla  $101\frac{1}{2}^{\circ}$ . When left foot was pricked she turned it up; pulse too rapid to count; heart's action tumultuous. Tr. digitalis, grs xv. 5 o'clock P. M. Spasm of lip still continues; lies on her back with eyes closed, and gives no evidence of pain when any part of the body is pricked; pulse in same state. 6 o'clock P. M. Breathing heavily; eyelids closed and eyes turned upward; pupils do not contract to light, but lids contract slightly when conjunctiva is touched; reflex irritability very much impaired; pulse 100; temperature  $102^{\circ}$ . 7 o'clock P. M. Spasm of mouth has ceased; respiration very slow and feeble; pulse 80; temperature  $102^{\circ}$ . 10 o'clock P. M. Mucous rales heard over whole chest. 12 o'clock A. M. Patient remains unconscious. 2 o'clock P. M. Patient still breathes slowly and feebly; small amount of frothy mucus comes out of her mouth; patient remained in this condition until death, 10 A. M., 9th instant.

*Autopsy.*—Head: dura mater normal; sinuses empty; moderate effusion into arachnoid cavity; pia mater intensely congested; left middle cerebral artery about  $\frac{1}{2}$  inch from its origin occupied by a firm thrombus; beyond this the artery was thin, ribbon-like, scarcely perceptible, and finally lost; membranes readily detached from the brain, leaving the sulci gaping widely over the under surface of anterior lobe, left side about third frontal convolution and island of Reil. In detaching the membranes portions of brain-substance were removed with them, leaving an almost pul-taceous mass exposed; indeed the whole of under surface of anterior lobe was much softened, but this was most marked near the lateral border; under surface of middle lobe slightly softened; superior and lateral aspect of anterior and middle lobes from fissure of Rolando forwards was in a very softened condition, breaking down under the least pressure, of a pale yellowish-gray color, in marked contrast with other parts of the brain, which on section showed very numerous puncta vasculosa, and were of the normal color. Thalamus opticus somewhat softer than that of the right side; corpus striatum much softened and of a yellowish color. Thorax: lungs oedematous, and poured out an abundance of mucus on section. Heart: insufficiency of mitral valve; no vegetations noticed; left ventricle entirely filled by a firm white clot entangled in chordæ tendinæ and projecting into aorta; abdomen, kidneys, liver, and spleen much congested.

**Causes.**—Men are more often subject to arterial thrombosis than women or children, though we find the great number of cases of thrombosis of the sinuses to be among women, and this perhaps due to the tendency of this sex to chlorosis.

Gintrac considers very young children to be subject to venous thrombosis. Of 37 cases seen by him, 14 were among infants; but arterial thrombosis is a condition peculiar to advanced life, and instances before middle age are not at all common unless they be of a specific nature. The exciting causes are numerous, but it may be assumed in nearly every instance that the blood is in a state of hyperinosis as a consequence of acute disease, such as rheumatism or pneumonia. Excessive heat is very often a cause. Dickinson<sup>1</sup> gives four cases, in two of which heat was the cause, in one other intemperance, and in the fourth violent vomiting,

In many of these patients there is old heart disease with some enfeebled action of that organ. The basilar artery, which receives its blood from the vertebral arteries, may be the seat of a clot at its remote end when heart force is preternaturally weak, but this is a rare form of the disease. I have already spoken of peripheral phlegmatous troubles, and it is only necessary to call attention to the danger which may arise from carbuncle. The puerperal state favors the formation of thrombi, and just as phlegmasia alba dolens is brought about, so may the thrombosis of the cerebral arteries be produced. The graver variety of intracranial thrombosis may be produced by internal or external cause. Lancereaux collected 89 cases, 30 of which were connected with caries of some of the cranial bones, and 24 with otitis. In one-half of these cases there were multiple abscesses of the brain.

In conclusion I would allude to the possibility of traumatic origin, a variety of blood-states, and pressure from intracranial tumors, exostoses, and thickened meninges.

**Morbid Anatomy and Pathology.**—Von Dusch, Parnum,<sup>2</sup> Grissolo,<sup>3</sup> Zahn, and a host of observers have devoted themselves to the study of this subject, and since the original observations of Kirkes<sup>4</sup> were published in 1852, which were devoted to the pathology of thrombosis as well as embolism, a great deal has been written. Parnum and Burrowes<sup>5</sup> both experimented by injecting substances into the circulation, and Burrowes probably relates the earliest case of recognized thrombosis.

Zahn gives the following concise description of the pathological process which attends the production of the thrombus. "The intensity and the duration of the injury, together with the previous condition of the individual, determine the durability of the clot. The process of

<sup>1</sup> Loc. cit.

<sup>2</sup> Virchow's Archiv, xxv. 3—6, pp. 308—328, 433, 530, 1862.

<sup>3</sup> Pathol. Intern., p. 247.

<sup>4</sup> Med. Chir. Trans., 1852.

<sup>5</sup> Med. Gaz., vol xvi. 1834-5.

formation is the following. Colorless blood-corpuscles adhere to a part of the intima denuded by an injury of its endothelium. They accumulate there, form a ring-like obstruction, and gradually the clot obstructs the vessel altogether. If the injury be slight, and the nutrition of the individual unimpaired, the current of blood soon breaks through the blood-clot and carries along the flakes of the colorless blood-corpuscles. The normal condition is soon restored. If the injury of the vessel be more severe, and the surrounding tissue already in a state of irritation, the thrombus, whilst forming in the same way as described, is firmer and larger. The obstruction is more complete, and lasts for twenty-four hours and more; after that period the thrombus begins to disintegrate into granular fibrine, the outlines of the blood-corpuscles composing the thrombus cease to be visible, and thus an uninterrupted circulation is re-established."<sup>1</sup> In more serious trouble the detached clots may be the nuclei of larger ones in the sinuses if the condition of the arterial walls be such as to favor more extended formation of thrombi so that the vessels become entirely occluded.

The consequence of arterial occlusion is the formation of an extended clot which blocks up the vessel more fully, and consequent ischæmia of distal parts. Through the agency of outside vessels collateral circulation is generally established in a short space of time. If, however, the anatomical site be such as to interfere with this provision of nature, softening or tardy degeneration will ensue. This softening, when it follows, is expressed by a series of changes, which occur about as follows: Red softening in from 24 to 48 hours, while the yellow change does not take place until after 14 days. But of this condition of affairs I will speak in a subsequent chapter. The carotid arteries and their termination are more often affected, and basilar vertebrals, anterior cerebral, and posterior communicating come next, in the order that I have given them. The pathological processes in the second form of intracranial thrombosis, viz., that affecting the sinuses and veins, are much more gross. Either through sluggish circulation of the blood on the part of a weak heart, pressure upon a sinus, or unusual density of the blood, coagulation occurs, the arterial flow is interfered with, a part of the brain is deprived of blood, and serum is effused. If the disease be due to outside causes, there may be an extension of inflammatory action from without in the manner I have described. By an extension of thrombosis, a form of meningitis resembling tubercular meningitis may be produced. Several of these cases have been seen by Scuch.<sup>2</sup> An artery which is the seat of a thrombus presents these appearances:—The inner coat is rough and perhaps corrugated; the artery as a whole may be hard and discolored, with diminution in calibre and a deposition of recent or ancient date, in which latter case it will be pale and tough, while atheroma is not un-

<sup>1</sup> Virchow's Archiv, Band lxii., Heft 1, Nov., 1874.

<sup>2</sup> Verhandlung der Wurzburg. Med. Gesellschaft, viii. 179.



commonly present. Fox<sup>1</sup> has observed that the part of the clot adherent to the inner coat of the vessel is much more dense than that nearest the centre. When the capillaries are implicated, they are generally found to be hard and calcareous. In thrombosis of the large sinuses or veins, the morbid appearances are much more striking. The thrombi are large, and, if old, of a gray color, and it is not rare to find pus-effusions of serum into neighboring parts, and perhaps some meningitis. Von Dusch has collected 57 cases, which are given by Fox.<sup>2</sup> In 32 the thrombosis resulted from gangrenous, erysipelatous, and other inflammations of the body (chiefly of head). In 15 it appears to have resulted from asthenic circulation. In 6 cases nothing positive could be ascertained.

**Diagnosis.**—There are very few conditions with which that under consideration may be confounded. When we remember that in thrombosis the development of symptoms is gradual, the loss of speech incomplete, and primary; and in cerebral hemorrhage the onset is sudden, the aphasia is secondary to a loss of consciousness, and the paralysis more marked, the diagnosis from this disease is not so difficult. Doubts may arise in our minds when we are to decide whether or not the case before us is one of thrombosis or uncomplicated softening. Thrombosis is rarely attended by marked elevation of temperature, while the opposite is to be observed in cerebritis, which presents as symptoms trembling and perhaps muscular rigidity. The psychical symptoms are also more strongly marked. The more serious form can be diagnosed by the coexistence of other conditions which may favor its origin.

**Treatment.**—The chief indication seems to be: The improvement of the condition which influences the production of the thrombus. If arterial tension be at all weak, we may combine digitalis and iron, give tonics and improve the patient's general condition by good food and stimulants. Nature will arrange the process of collateral blood-supply, and we may aid her by enforcing rest and quiet.

## THROMBOSIS OF SINUSES AND VEINS.

When a large sinus or vein is involved, the resulting symptoms are much more complex and difficult to diagnose.

Lancereaux,<sup>3</sup> who has written quite extensively about this form of disease, has divided into two grades, in regard to the variety of morbid action. One of these is inflammatory, the other is non-inflammatory. The first form is dependent upon the extension of some inflammatory process, usually from the ear, while the other is attended by coagulation of the blood in sluggish circulation.

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<sup>1</sup> Path. Anat. of the Nervous Centres, p. 32.

<sup>2</sup> Loc. cit., p. 35.

<sup>3</sup> De la Thrombose, etc., Paris, 1862.

Von Dusch<sup>1</sup> does not agree with him, but Tonnelé, quoted by Grisolle,<sup>2</sup> makes the same varieties as Lancereaux.

The seats of this pathological condition are the longitudinal, lateral, basal sinuses, and the large veins communicating therewith. Bastian<sup>3</sup> alludes particularly to the longitudinal sinus as the most common seat and describes the tendency to plugging up of the cerebral veins on both sides.

As I have said, the symptoms are very obscure, but in every case we may consider them to be the indication of pressure. Headache, delirium, coma, convulsions, ocular troubles, and generally death in a very short space of time mark the course of the disease. Mr. Tuckwell<sup>4</sup> reports a case which is a representative of the anæmic form. It is as follows:—

Eliza C., æt. 16, was admitted to Radcliffe Infirmary on the 20th day of April, 1871. She ceased working a month before on account of palpitations, shortness of breath, weakness, irregularity of the menses, etc. Two weeks before admission she began to suffer from violent headache. She never had fits. A condition of decided chlorosis was diagnosed. There was a systolic murmur at base and venous murmur in the neck; nothing else abnormal was detected. She was put to bed.

*April 21.* She sat up, but it was noticed that she lolled about in a strange manner, and seemed stupid. Her right hand and arm were weak, and she could not raise them to shake hands. Headache still severe.

*24th.* Remained in same apathetic state; the paralysis of arm had increased, and she could not move fingers or hand at all; headache. She became comatose, and died after the visit of Dr. Tuckwell and his colleague, Dr. Palmer.

*Autopsy* twenty-four hours after death. On removing skullcap, the dura mater covering right hemisphere was found to be of a dark color, and the longitudinal sinus, when examined, was found half way blocked up by a firm white blood-clot of some age. Cerebral veins on the surface of the middle and posterior part of right hemisphere were all occluded by dark clots. On removing the brain, blood was found effused in the right middle cerebral fossa, extending down into the spinal canal.

Lateral and basal sinuses were filled with clots of some age. The pons and medulla were covered by a clot of recent date. General softening of the brain was observable, the optic thalami and corpora striata being particularly affected. The arteries were all healthy, as well as the bone about the sinuses.

Another case is reported by Dr. Tuckwell, which presented symptoms which were very much like those of his own case.

Von Dusch<sup>5</sup> has spoken of epistaxis with thrombosis of the longitudinal sinus as a common symptom, and Meissner has called attention to grinding of the teeth, profuse diarrhœa, and exhaustion, together with certain changes in the configuration of the head. In children he has found de-

<sup>1</sup> Zeits. für Ration. Med. B. vii., 1859, p. 11.

<sup>2</sup> Op. cit., tome ii. p. 240.

<sup>3</sup> Paralysis from Brain Disease, etc., p. 22.

<sup>4</sup> St. Bartholomew's Hospital Reports, vol. x., 1874, p. 35.

<sup>5</sup> Loc. cit.

pressed fontanelles, lapping of cranial bones, and unequal distension of the jugular veins. Metastatic abscesses, indicated by local symptoms, have been found by many observers. Lancereaux estimates that nearly half of all the cases are thus complicated. I have seen one case where erysipelas was undoubtedly the cause of the cerebral thrombosis, and after death the great sinuses were found to be filled with semi-purulent matter, and there were abscesses in the liver and other parts of the body. These cases are not so exceptional as they are generally supposed to be, but diagnosis before death is rarely made.

An autopsy made at the New York Hospital by Dr. Amidon, who kindly invited me to be present, revealed the following beautiful evidences of thrombosis of the cerebral sinuses which followed septicæmia :

The boy had died after several days' illness, the original injury being a compound fracture of the bones of the left leg. The autopsy was held on September 15th, the day of his death.

The liver, kidneys, and lungs showed evidences of acute congestion, and the heart contained two *ante-mortem* clots; one occupying the right auricle, and the other the right ventricle. The lungs were carefully examined, and a pyramidal infarction was found at the border of the inferior lobe of the left lung. The head was opened, and the dura mater was found to be quite healthy, except in the superior longitudinal sinus, which was almost completely filled with a well-organized thrombus of a pale color. One of the large descending veins in the parietal region was occluded, and when the dura mater was removed, a large pouch, filled with limpid and perfectly clear serum, was found beneath, which pressed upon the parietal convolutions just posterior to the fissure of Rolando. This was beneath the arachnoid. At no other point was there any abnormal collection of fluid, and in no place was there any evidence of structural changes of the brain-substance proper. The lateral sinuses were partially filled with thrombi, and contained some very fluid blood. The left petrosal vein was empty, as were others which were higher up. No arterial occlusion was found. The patient had died suddenly in convulsions with coma.

**Causes.**—Blows upon the head, injuries of various kinds, extension of otitis, intemperance, and the causes I have already enumerated, may be mentioned. There seems to be no special dependence upon age or sex, though it may be said that most of the cases occur during adult life.

What I have already said, and the excellent cases of Tuckwell, which have been presented, render it unnecessary to say more about the *morbid anatomy, pathology or diagnosis*.

In regard to the prognosis, there can be no question. It is about as bad as it can well be. As to *treatment*, the most we can do is to build up our patient, and reduce the danger of external disease by favoring a free escape of pus if the original disease be otitis, and there be an accumulation. We may employ local cold and derivatives, but even these do little good after the disease is recognized.



## EMBOLISM OF THE CEREBRAL VESSELS.

The cerebral arteries and capillaries are alike subject to this form of mechanical obstruction, but the former are perhaps the most common seat of the lodgment of fibrinous plugs. The little bodies which are forced into the vessels are always from some other part of the system, and are not formed in the vessel, as is the case in thrombosis.

Embolism also differs from thrombosis in the fact that the latter is always developed, and attended by gradual narrowing of the vessel; while the condition under consideration is a sudden accident, and may occur in a perfectly healthy vessel; the converse is the rule in thrombosis.

**Symptoms.**—Unless there is previous acute endocarditis, there will seldom be any warning, the patient being suddenly stricken down as the little plug is violently forced into some vessel of the brain. There may even be no loss of consciousness, though this is the exception. Unconsciousness invariably occurs when a large embolon plugs up some such artery as the middle cerebral; but if the embolon be small, and the artery occluded is one concerned to a very limited extent in the vascular supply of the cerebrum, the unconsciousness may be but transitory, and psychical symptoms of slight moment will constitute the sole indications of confused mental activity.

The eyes are sensitive to light, the pulse is small and rapid, and there is usually pallor. There are no indications of pressure, no stertor, no tumultuous respiration, nor full pulse, and the pupils are either dilated or irregularly contracted.

If the heart be auscultated, various murmurs or friction-sounds will in many cases be heard. Mitral murmurs are perhaps the most common.

Paralysis taking the form of complete or incomplete hemiplegia is the result of such sudden arterial occlusion.

Special facial muscles may be those affected, or various modifications of sensation, such as anæsthesia or hyperæsthesia, may be detected, but rigidity or contractures are rarely present unless there is secondary disorganization, and they are never seen during the early stages. Vertigo is a disagreeable and common symptom, and is sometimes attended by cerebral vomiting. Of course aphasia is an almost invariable consequence of embolism, as the middle cerebral artery is so commonly occluded. This aphasia is of variable extent, and is ataxic or amnesic, but generally the latter. On the other hand, the patient may be simply stupid and taciturn, refusing to answer, or he may be troubled with a light form of clumsiness or slowness of speech. The headache, which is subsequent to the loss of consciousness, is coincident ordinarily with the re-establishment of collateral circulation, and if further changes occur there may be intense head-pain, delirium, mania, or symptoms indicative of softening. The duration of this stage varies greatly. I have seen examples where the symptoms were trifling and transitory, such as headache, awkward

speech, and paralysis of one arm rapidly disappearing. Other cases are correspondingly serious. Mr. Shaw<sup>1</sup> reports a case which proved fatal in twenty-four hours, and others have detailed examples in which death ensued in from thirty-six to forty-eight hours.

It is very common to find, at the same time, symptoms indicative of embolism of other organs. The spleen, lungs, and organs which receive a large supply of blood, or are in the direct line of arterial supply, are apt to be involved as well as the brain. It rarely happens that two or more cerebral arteries are simultaneously plugged. In such cases the symptoms are complicated. One case is recorded in which both middle cerebral arteries were occluded, and the following case reported by Sokolowski<sup>2</sup> is an example of coexisting splenic and cerebral embolism:—

The patient was a servant, married, aged 23, who had always menstruated regularly, except when she was pregnant second year before, and then gave birth to a healthy child. Her health had been ordinarily good. Four days before her admittance to the hospital she had suffered from alternate chills and heat, with headache and constipation. On admission her pulse was 100; temperature, 102.6°. Heart friction sound at apex, but nowhere else. Passed 53 oz. urine in 24 hours; sp. gr. 1025.

*October 13th.* She suddenly became paralyzed on the right side, lost all power of speech, and only moaned and cried in a frightened manner. The third day after, acute idiopathic endocarditis was diagnosed. The right ventricle was found to be greatly enlarged. Temp. 101.2°; pulse 100. After paralysis she lost hearing in the right ear; pupils were normal; left side of mouth was drawn up. Anæsthesia of paralyzed parts. Urine and feces passed unconsciously. Spleen tender and enlarged. An additional diagnosis was now made. Embolism of left middle cerebral artery, and embolism of splenic artery. The loss of speech was peculiar. She was unable to articulate at all, though there was sufficient evidence of mental activity and originating power, so she communicated with her friends by signs. The paralysis had begun to disappear in the right leg below the knee, and she could move her foot slightly. The temperature on the first day was 102.2°; pulse 90. In the evening, 104.8°; pulse 100. On the second day, Oct. 14, there was much improvement. The morning temperature was 102.8°, and the evening 103.8°.

*15th.* All paralysis and alalia have vanished. She is, however, extremely weak. During the next two or three days a diarrhœa, loss of appetite, and considerable increase of tenderness over the spleen appeared.

*28th.* 35 oz. of urine were passed, which contained albumen, hyaline casts, and urates in abundance.

*November 10th.* She has grown gradually worse, is no longer able to answer questions, but repeats words and sentences over and over. There is marked loss of memory. The fever has greatly increased, the evening temperature being 105.2°; pulse 120, and quite thready. There are evidences of bronchitis and pulmonary difficulty. Urine greatly decreased in quantity, and albumen increased; tongue quite dry.

<sup>1</sup> Trans. of Path. Soc. of London, vol. iv.

<sup>2</sup> Deutsche Med. Woch., Dec. 15, 1875.

20th. She died. There was extensive hypostatic pneumonia; consciousness remained to end.

*Autopsy.*—Arteries at base healthy, except middle cerebral on left side. This contained a semi-transparent embolism of cartilaginous consistency. Right side of brain healthy, though pale. The left side in the same condition, except at the island of Reil, and gray matter of lenticular nucleus, which were small, hard, and yellow, and showed evidences of softening and subsequent cicatrization. The heart was enlarged, and yellow spots were found beneath the endocardium. The edges of the mitral valves were thickened and covered with coagula. The spleen enlarged, "blocked," and the splenic artery occluded.

Cases have been reported where embolism followed, or was connected with, chorea, and this connection has been made use of in the explanation of the pathology of the latter disease. One of these cases, seen by Murchison,<sup>1</sup> is worthy of mention.

The patient, a boy 14 years old, had suffered from chorea when seven years old, from which he recovered. Two weeks before he died, irregular choreic movements appeared, connected with a bellows murmur at the left apex. When seen, June 12th, the pulse was 120; temperature 102°. There was a pericardial friction sound, but no pain in joints or other symptoms of rheumatism or endocarditis.

June 28. Sudden unconsciousness, head drawn to right side, extreme rigidity, twitching on right side. Pulse 145. Pupils normal and equal, but subsequently contracted; no paralysis. Died June 29. Vegetations on mitral valves, spleen containing emboli. Left vertebral and left internal carotid arteries blocked by pale, firm, and easily detached coagula; left hemisphere considerably softened. Examination revealed no small emboli in capillaries.

A case of my own, showing an accident which may occur in the course of certain acute diseases, seems to me to be of sufficient interest to present, as it may call attention to a cause of death which is probably sometimes overlooked.

Mr. N., æt. 35, a stout, full-blooded man of good habits and no vices, took to his bed on the 25th of June, 1874.

He had contracted a "bad cold" at the theatre, and the next day was seized with pain in the left side, was chilly and uncomfortable, and when I saw him on the evening of the same day, he had a violent headache. His skin was hot, and his pulse hard and rapid. The thermometer indicated a temperature of 101°; pulse 122. At the base of the left lung crepitant râles were heard. Flaxseed poultices were applied, and quinine and other remedies administered. For the next four or five days the lungs underwent consolidation, and nearly all of the physical signs connected with the different stages of pneumonia were observed. The most marked of these was a high temperature, which ranged between 103° and 105° for six days. Resolution was slow, and but a few sputa were brought up, but the temperature had fallen to some extent. I was sent for in haste on the evening of the fourteenth day, an hour after my ordinary visit, to

<sup>1</sup> London Path. Soc. Trans., vol. xxii.



find that the patient had suddenly, while taking his beef-tea, fallen back unconscious, and had remained so ever since. This was about half an hour before my being sent for.

His pupils were widely dilated, and his corneæ when touched were sensitive; his legs and arms were extended. His temperature was not high, and his breathing had not changed very much from what it was when I saw him earlier in the day.

After an hour and a half he made some movements which showed slight voluntary control, and vomited, turning his head slightly to do so. He uttered no sounds except low moans. Towards morning his breathing became more troubled, and he rolled in the bed.

At about nine o'clock in the morning of the next day he seemed to recognize those about him, and made signs which were not understood, when he knit his brows and seemed perplexed. He refused food, but permitted an enema of beef-tea to be injected, but this was not retained. It was then found that he was hemiplegic on the right side. Later in the day he passed his urine in bed.

*16th day.* Did not sleep last night. The temperature  $104^{\circ}$ ; pulse, 130, full and hard. After my visit this morning he became comatose. 3 P. M., died.

*Autopsy* 20 hours after death.—Lungs: right, rather more pinkish than normal; some spots of induration at base. Left, solidified throughout most of its substance; when cut, bloody serum exuded. Heart somewhat enlarged. Mitral valves were covered by stringy clots. The right ventricle contained a large fresh clot. Kidneys: right, normal; left, somewhat smaller than it should be; contained a small cyst beneath the capsule. Head: On opening the cranial cavity, the vessels of the dura mater were filled with dark blood. The longitudinal sinus contained a quantity of thick, clotted blood, which was almost black. The left hemisphere was œdematous, except at a point beneath the lateral ventricle, where there was a circumscribed patch of a pinkish hue, which seemed to be well defined. The left middle cerebral artery, at a point just before it gives off its branches, was found to be swollen and hard, and when cut open a small, rather firm clot was found. Behind this there was a long, stringy clot of more recent date. About the vessel the brain was œdematous. Another patch of red softening was found in the same hemisphere somewhat more posteriorly. No other large arteries were affected, but when microscopically examined, I found considerable occlusion of many small capillaries, and great disorganization of the nerve element.

I have seen several other cases of this kind occurring during acute diseases attended by a hyperinosed condition of the blood.

**Causes.**—Endocarditis is, above all other causes combined, the most important and common in the production of embolism. At the Pathological Institute of Berlin<sup>1</sup> there were 300 cases of embolism of all kinds associated with endocarditis during the years included in the period beginning 1868, and ending 1871. Twenty per cent. of these cases were of brain embolism. Of a large number of cases reported in the London Pathological Society's Transactions, nearly all of them were of this

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<sup>1</sup> Edinburgh Med. Journ., July, 1873.

nature; and out of fifteen cases I have seen, twelve were connected with disease of the heart, and generally with deposits upon the mitral valves.

Croup, the puerperal state, phlebitis, and other conditions where there is any tendency to the formation of clots, or the detachment of tissue which finds its way into the circulating apparatus, may all produce embolism.

Numerous accidents which happen through carelessness, or perhaps unavoidable injury during surgical manipulation, may, by the introduction of a blood-clot or foreign substance into the circulation, produce an occlusion of some cerebral or other vessel. This accident has occurred when pressure has been made upon large aneurisms, and is one of the arguments against the intravenous injection of substances which coagulate the blood, such as ergot, persulphate of iron, hair, or other organic substances.

Dr. Barker<sup>1</sup> has given two cases of embolism following the parturient state, and Thomas has seen one or more cases of this kind.

As to age, I have found that more young people have had cerebral embolism than persons of advanced life. An examination of twelve cases reported by different observers gives the relative frequency as follows :—

|                                   |   |                                   |   |
|-----------------------------------|---|-----------------------------------|---|
| Between 10 and 20 years . . . . . | 2 | Between 40 and 50 years . . . . . | 2 |
| “ 20 “ 30 “ . . . . .             | 4 | “ 50 “ 60 “ . . . . .             | 1 |
| “ 30 “ 40 “ . . . . .             | 3 |                                   |   |

Of these, 3 were males, and 9 were females.

Of my own cases, seven were between twenty and thirty; five between thirty and forty; and three between forty and sixty. Eight were women, and the others men. It seems, therefore, that the period between the twentieth and thirtieth years is that in which the disease is most common, and that women are most subject to the disease. According to the observations of medical writers in general, mitral disease is more often an affection of youth or early life than of advanced years; so it seems probable that people who have not reached middle life should be more subject to embolism.

**Diagnosis.**—The important distinction is to be made when we suspect the case to be one of cerebral hemorrhage. Next in order come thrombosis, cerebral congestion, meningeal hemorrhage, and cerebral tumor.

Gelpke<sup>2</sup> has given the following table, on one side of which are detailed the features of cerebral embolism; on the other, those of cerebral hemorrhage :—

#### CEREBRAL EMBOLISM.

Youth of patient.  
Sudden onset without prodromata.

#### CEREBRAL HEMORRHAGE.

Advanced age, atheroma.  
Prodromata generally present.

<sup>1</sup> Puerperal Diseases, p. 270.

<sup>2</sup> Archiv der Heilkunde, xvi., Aug. 1875, p. 485.

Previous articular rheumatism, valvular sounds.

Previous disease, which might lead to formation of clots.

Hypertrophy of left ventricle.

*The Attack.*

Extensive muscular paralysis ; amnesic aphasia.

Very rapid ; or quite imperceptible disappearance of the residual disorder.

Retention of early mental power.

*The Attack.*

Symptoms of cerebral pressure ; ataxic aphasia ; involvement of the intelligence.

Disappearance of the residual disorder after a moderate time.

Reaction stage.

Janeway<sup>1</sup> relates an admirable case to illustrate the obstacles sometimes encountered in making a diagnosis. As it will be seen in his case, there were many circumstances of a puzzling character which made the diagnosis exceedingly difficult.

A young woman, while at work, fell to the floor unconscious, in what appeared to be a "fainting fit." There were some convulsive movements limited to the left side of the body. When admitted to Bellevue Hospital on the following day, there were irregular contraction of the pupils, coma, and high temperature. A loud systolic murmur was heard all over the chest. She remained unconscious for two days, and on the third day died. Her breathing previous to death was stertorous, her limbs flaccid, and reflex action diminished. The pupils were dilated. Her urine contained a small amount of albumen, but not enough, in the absence of œdema and other symptoms, to suggest nephritic trouble ; besides, the quantity of urine passed was sufficient. The question of thrombosis was excluded by the absence of premonitory symptoms. Congestive chill was suggested by the paralysis and meningeal hemorrhage, but excluded when the absence of rigidity was taken into account. Janeway considered the lesion to be hemorrhage, and I will give his own description of the autopsy and its result.

"The *post-mortem* examination revealed the following: Skull, normal. Brain and membranes: On opening the dura mater on the right side, a clot of blood, a little over half an inch thick, three inches long, and two inches wide, escaped from the arachnoid sac. This clot was in the main, black, moderately soft, but provided with a buffy coat at one portion. It had produced a corresponding depression of the brain, over which it was situated, and in its centre was an opening about an inch long and a half inch wide, leading from a recent excavation in the middle lobe of the brain, through the torn pia mater and so-called arachnoid, into the sac of the latter. This excavation reached from the convex surface nearly to the corpus and optic thalamus at posterior extremity. The opening was situated a little nearer to the longitudinal fissure than would correspond to the middle of the convex surface. The excavation was about two inches wide and contained clotted blood, of which some had escaped in removing brain. The brain-tissue surrounding this was soft, slightly blood-stained, and where it formed the boundaries of the space,

<sup>1</sup> Am. Psychological Journal, Nov. 1876.



numerous small torn vessels. The brain-tissue of the posterior lobe, especially on its outer surface, was softer than natural. The posterior extremity of the optic thalamus of the right side, over a small area, presented an ecchymotic softened state.

"In the clotted blood and disintegrated brain-tissue found at the mouth of the excavation, a small branch of the posterior cerebral was found torn across, presenting a widened extremity at the point of rupture, surrounded by thickened and firm tissue, and in the interior of this a firm reddish-gray clot, uniform in its structure and of older date than any others. I failed on careful examination to find the other extremity of the torn vessel, but from the condition of the portion found doubt not that it would have proved of similar shape to the other, and that together they would have constituted a cylindrical dilatation of this artery.

"The left (opposite) hemisphere showed the convolutions flattened and so closely pressed together laterally as to nearly obliterate the appearance of sulci. The arachnoid was dry, and there was no sub-arachnoid fluid present. The brain on this side appeared anæmic, and on cutting the dura mater, pressed out.

"The lateral ventricles were of normal appearance. The anterior lobe of right side was normal. Pons, cerebellum, etc., were normal. The arteries at the base were carefully examined, being followed to their smaller ramifications without finding any emboli.

"The lungs were slightly cedematous.

"Heart: The left ventricle was slightly hypertrophied. On the auricular aspect of the mitral valve, and on the ventricular of the aortic, condylomatous excrescences were present, narrowing both orifices; but the largest mass passed obliquely across the heart from the leaf of aortic valves nearest the septum to the anterior leaf of mitral valves, and above this, between it and the other leaflet of aortic valves, a slight dilatation of the heart-wall existed.

"Small infarctions were present in the spleen and the kidney, and the latter showed at some points interstitial nephritis, around glomeruli, with atrophy of these; but the disease was not advanced. The mesentery presented two small aneurismal dilatations of little arteries, and at these points emboli were present: one was of the size of the head of a pin; the other, of a pea.

"In this case it seems exceedingly probable that the primary lesion of the artery, which finally ruptured, was embolism, and that this obstruction caused, secondarily, a dilatation of the artery at this point, and that, owing to the heat,<sup>1</sup> such an obstruction of the circulation in the brain occurred as to cause the rupture of the vessel described. This is rendered still more probable by finding two small arteries in the mesentery with aneurismal dilatation, and containing emboli.

"A point of interest in this case is the absence of serious symptoms of cardiac disease, though there was so marked a lesion. It did not seem as if any regurgitation had occurred at the aortic orifice, simply obstruction. The left ventricle contained such a firmly adherent clot that the hydrostatic test was of no avail.

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<sup>1</sup> The weather was excessively warm at this time, and the patient was at first supposed by those around her to be suffering from the effects of the heat.

"It also furnishes another to the already long list of cases in which a heart-murmur is heard—sudden paralysis occurs—the patient moderately young, and yet the lesion is hemorrhage, and not embolism. I have met with several of these exceptions."

From thrombosis there will be no difficulty in making a diagnosis when we remember the slow origin of the former. The "apoplectic form" of cerebral congestion sometimes resembles the condition presented by the patient; however, the former history, the suffused face, contracted pupils, and rapid subsidence of symptoms, will put us on our guard.

**Morbid Anatomy and Pathology.**—Burrowes and Kirkes were the first English writers and Virchow the earliest Continental writer to describe these conditions. Prévost and Cotard have since related interesting experiments. They injected tobacco seed into the carotids of dogs, and afterwards watched the changes that followed. One of these dogs was killed thirty-nine days after the seed had been introduced, when they found the middle cerebral artery obstructed, and induration about the fissure of Sylvius.

The pathological processes which follow such mechanical obstruction have been sufficiently noticed in a preceding article, so it will be enough to call attention to the fact that the consequence of such an accident will be softening of the parts deprived of their nourishment, unless the collateral circulation be established at an early date, or the embolon is broken down and removed, which is a very unlikely circumstance.

Kirkes<sup>1</sup> calls attention to the distribution of emboli in the following words: "The parts of the vascular system, within which these transmitted masses of fibrine may be found, will of course depend in a great measure upon whether they proceed from the right or left side of the heart. Then, if they have been detached from either the aortic or mitral valves, they will pass into the blood propelled by the left ventricle into the aorta and its subdivisions, and may be arrested in any of the systemic arteries or their modifications in the various organs, especially those which, like the brain, spleen, and kidneys, receive large supplies of blood directly from the left side of the heart. If, on the other hand, the fibrinous masses are derived from the pulmonary artery and its subdivisions within, the lungs will necessarily become the primary if not the exclusive seat of their subsequent deposition."

In regard to the side of the brain where the deposit occurs, I think we may say that the left side and the middle cerebral artery are the most common site, though many cases reported by Shaw, Glynne, Murchison, and others prove that the right artery may be affected as well.

An interesting example, which is almost unique, is the following case of embolism of the right posterior cerebral artery. The history was read by Broadbent before the London Clinical Society:—<sup>2</sup>

"The patient, a young man aged 19, had suffered three years pre-

<sup>1</sup> Royal Med. Chir. Trans., vol. xxxv., p. 281, 1852.

<sup>2</sup> Abstracted from *Lancet*, Monthly Abstract, April, 1876, p. 576.

viously from acute rheumatism. Ten days before his admission, he suddenly became blind, and had great pain in the head. Five days later, vision having returned, he lost the use of his left limbs, while the right arm and leg were continually in motion; and, unless restrained, he rolled over and over towards the left, falling out of bed and bruising himself severely. The left hemiplegia and uncontrollable movements of the right limbs continued when he was admitted; the hemiplegia not being absolute, but accompanied by slight rigidity and very considerable impairment of sensation. The patient took no notice of persons or objects, but answered questions, and put out the tongue on being urged. His pulse was variable, 120 to 160 or more. Temperature in the right axilla, 99.2°; in the left, 100.6°. A loud mitral systolic murmur was present. The bowels were confined, and, when opened, the feces and urine were passed in bed. A dose of three grains of calomel was given, and two grains of carbonate of ammonia, with two drachms of infusion of digitalis every two hours. Chloral also was given at night. He was ordered a diet of milk and beef-tea, with four ounces of brandy. There was gradual improvement; and three days after his admission, an ophthalmoscopic observation, previously attempted in vain, was obtained, and the disks were found to present the appearances of marked ischæmia. The pulse was now 108, soft, short, and strikingly microtous. A day later the pulse was 88, and more full. The temperature was still nearly a degree higher in the left (100°) than in the right (92.2°) axilla. Slight paralysis of the left external rectus of the eye was observed. At the end of a fortnight's stay in hospital, the right limbs were quiet, and there was considerable return of power and sensation in the left side. His speech was rather slow, but there was no obvious impairment of the intellect. Notwithstanding this, however, he not only passed his feces in bed, but threw them about and bedaubed himself and the bedclothes without any regard to decency. The optic ischæmia was marked, but vision was good. The temperature of the right axilla was 99.3°; of the left, 100°. At the end of three weeks he passed his excretions naturally. After five weeks he was up and about, eating well; but pale, and still complaining a little of headache. Impairment of power and of sensation in the left limbs was still perceptible. The optic neuritis was subsiding. Distant vision was good, but small print was not easily read. A systolic mitral murmur was heard. The temperature was still never below 99°; usually 100°; it was now equal on the two sides. But for this elevation of temperature, the patient would have been allowed to leave the hospital. Soon afterwards, however, there were symptoms of splenic embolism, and later of ulcerative endocarditis; and he died from this four months after admission. On *post-mortem* examination, with ulcerative endocarditis and numerous recent embolisms, there was found softening of the occipital lobe of the right hemisphere from the posterior cornu of the ventricle downwards, and the branch of the post-cerebral artery entering the calcarine fissure was occluded and lost in adhesions. It was considered probable by Dr. Broadbent that originally the posterior cerebral artery itself had been blocked up, and not only this branch. The interesting points in the case, on which comments were made, were the temporary blindness, the agitation of the right limbs, and rolling tendency, the usual association of loss of sensation, and of double optic ischæmia with embolism of a cerebral artery, and the remarkable indifference to decency persisting when the intellect was apparently good."



Fat globules may sometimes plug up the small capillaries, producing wide areas of softening.

The morbid appearances indicative of cerebral softening are of interest and worthy of the closest study, not only because the brain is the point which suffers the most seriously, but because generally the heart, spleen, lungs, blood-vessels, and other organs may be involved as well. On the valves of the heart, either mitral or aortic, may be found excrescences, induration or recent clots, and the arteries themselves may exhibit patches of atheroma. In the brain we will probably find one or more of the arteries I have spoken of to be swollen, hard, and filled by one of these little masses of fibrine. They have been compared to grains of wheat, and resemble them very closely. Generally the embolon is separated from a second plug which has followed clotting of the arrested blood. Emboli are never *attached* to the walls of the vessels.

Several arteries may, perhaps, be found obstructed in the same way. "Sometimes all on one side; at other times some arteries of one side of the brain, and some of the other,"<sup>1</sup> so says Fox.

Softened masses are generally found on examination, and are usually the cause of death. The parts behind the occlusion are subjected to the full force of blood which is arrested, and not sent to the parts it should supply, and local hyperæmia is a result. The resulting softening is generally confined to the left hemisphere at its base, for reasons I have before stated, and the frontal convolutions, corpus striatum, and adjacent parts are found to be either red or yellow, softened or indurated.

Oedema of the brain is not an uncommon appearance, such oedema being seen in the parts deprived of blood. The perivascular spaces being enlarged, it is but natural that their fluid should rush in to fill up the increased space left by the bloodless arteries.

**Prognosis.**—The outlook for the patient is generally a very gloomy one if the accident be at all grave, and the artery be one of importance. The severity of the symptoms, the existence of emboli in other organs, the element of severe pain, high temperature, and gradual development of symptoms indicative of softening are of unfavorable import, and give affairs a very dark look; therefore it is never well to make too hasty a prognosis.

**Treatment.**—Rest, abstinence from stimulants, and agents which will diminish the arterial tension are the only remedial means to adopt besides the ordinary indications which appeal to the common sense and discretion of the medical man. Afterwards, resulting conditions, such as paralysis or softening, are to be treated.

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<sup>1</sup> Op. cit., p. 32.

## CHAPTER V.

## DISEASES OF THE CEREBRUM AND CEREBELLUM (CONTINUED).

## CEREBRAL SOFTENING.

**Synonyms.**—Remolissement (rouge, blanc, jaune). Encephalitis ägüe, chronique (Fr.). Mollities cerebri, Encephalitis, Softening of the Brain (chronic, acute), Inflammation of the Brain.

**Definition.**—A disease of the brain attended by destruction of nervous substance, and either of an acute inflammatory nature, with purulent formation; or of a chronic non-inflammatory character, with less rapid disorganization of nerve-tissue.

So much confusion has arisen from an incorrect appreciation of the morbid anatomy and its connection with pathology, that it is a difficult matter to attempt there conciliation of the many widely differing views of the legion of writers “Inflammation of the brain” is the term which has led to all this confusion; and I have been bold enough to base my classification rather upon the character of tissue-changes than upon the arbitrary law that softening of the brain is the only result of inflammation. Sclerosis, as we know, is undoubtedly the result of a low grade of inflammation, but in this case the tissue-changes are quite different.

Considering that the word “softening” means a mollification, and that it may result not only from purulent inflammation, but from low nutritive changes, I shall divide the subject as follows:—

- |                       |   |                      |
|-----------------------|---|----------------------|
| 1. Acute Softening,   | { | Diffused Cerebritis. |
| (Inflammatory),       |   | Meningo-Cerebritis.  |
|                       |   | Purulent Cerebritis. |
| 2. Chronic Softening, | { | Primary Softening.   |
| (Non-Inflammatory),   |   | Secondary Softening. |

1. Under the first head we may place the variety described by Elam,<sup>1</sup> which is a quite rare affection in its uncomplicated form, that is, when it involves the brain substance *en masse*; and meningo-cerebritis, which is by far more common. In a third variety the acute disease is characterized by purulent collections, and perhaps by the ultimate formation of abscesses.

2. Chronic softening in its primary form we will consider to be dependent upon general disease, intellectual prostration, and like causes;

<sup>1</sup> Cerebria, and other Diseases of the Brain, London, 1872.

while "secondary softening" may be used to express the form which follows vascular lesions, such as embolism, thrombosis, or cerebral hemorrhage.

## ACUTE SOFTENING.

In the first form it may be either cortical, diffused, or combined with meningitis.

**Symptoms.**—Cerebritis of either kind is preceded in nearly every instance by symptoms of functional disorder, such as cerebral congestion or cerebral anæmia, but these are not sufficient in themselves to arouse the suspicion of the observer as to the serious character of the disease which is to follow. The later prodromata of cerebritis, however, cannot be mistaken, and finally the developed disease presents most pronounced symptoms, which, if they do not always enable us to locate the brain lesion, are sufficient to assure us that some violent inflammatory process is under way in the cerebral mass. The patient may for some months suffer greatly from headache of a diffused character, accompanied by burning sensations, and a sense of pressure behind the eyeballs. These headaches are quite intense, and are aggravated by exposure to heat, concentration of the mental powers, and alcoholic indulgence. His memory becomes gradually enfeebled, so that at first dates and names are forgotten, and afterwards faces, locations, and even information which may have been imparted to him a short time previously. Some slight clumsiness of speech may be indicative of the near approach of grave symptoms, but this clumsiness is not aphasic till later. Irritability of temper, restlessness, and incapacity for mental application are attendant evidences of the smouldering fire which afterwards is to make itself known by still more decided symptoms. Among these may be enumerated nystagmus, strabismus, diplopia, and optic neuritis, as ocular troubles; contractures of the limbs, tremors of individual muscles, or groups of muscles, a twitching of the limbs, or other motor troubles, and hyperæsthesia, followed by anæsthesia, and other disorders of sensation; these last sometimes being peculiarly prominent. Next we find that there may be an apoplectic attack or convulsions of an epileptiform character, which mark the violent stages of the disease. Should there be, as a result of the morbid process, cerebral hemorrhage, it will be found that the paralyzed limbs become markedly contracted, and that rigidity is a striking feature, as the result of descending degeneration. According to Jaccoud, the contractures may be bilateral, though the rule is the other way, the limbs of but one side being rigidly flexed.<sup>1</sup> He has seen one case where the left arm and leg were the seat of contractures, and where the face was contracted and strongly drawn towards the left side, suggesting a right facial palsy, but the appreciable rigidity of the facial muscles of the left side left no doubt as to the origin of the deviation. The paralyzed members are generally

<sup>1</sup> *Traité de Path. Interne*, vol. i., Art. *Enceph. aigue*.



those that are the seat of convulsive movements in the first place. The convulsions may be general, and assume an epileptiform character, and may be accompanied by vomiting. The patient's mental condition meanwhile undergoes a great change. Delusions, which somewhat resemble those of general paralysis of the insane, are present; the *exaltation délirante* of the French, which is by some considered to be an early symptom. This has not been my experience, and I am convinced that in the cases where it has been noticed as an early expression of the affection, the disease was probably general paralysis, and not cerebritis. The real departures from mental integrity are expressed in a want of decision and a restlessness which is shown in the impaired fixedness of purpose. The patient repeats himself in conversation, and forgets that he has made the same statement but a few minutes previously. Memory is ultimately abolished, and finally dementia remains, which, should the patient live for some time, is expressed by all the other signs, drivelling of saliva, inane smile, hebetude, and total imbecility, while there may be aphasia of the amnesic or ataxic variety. The muscles concerned in articulation and deglutition are involved, and the patient may narrowly escape being choked by the masses of food which "go down the wrong way" or accumulate in his mouth. Constipation and retention of urine are not uncommon accompaniments, and the urine is charged with urates, is dark-colored, and rapidly undergoes decomposition. The temperature and pulse are both changed, the latter becoming accelerated and irregular, and the heart-sounds sharp and "precipitative." A tremulous character of the pulse has been noticed by several observers. The temperature may rise to 110° F., and generally attains its highest point at the end of the first four days. Coma precedes a fatal ending in the acute form at the end of a few days, and death occurs generally after seven or eight days by asphyxia. Should the patient survive, there is a remission of the symptoms, and the formation generally of an abscess. Cerebritis does not always begin in the same way, and, as I have already stated, is not invariably symptomatized by all the forms of disordered function I have enumerated. There may be no premonitory symptoms should the disease follow otitis or injury, but in the insidious form, which has been so admirably described by Elam and Reynolds, the appearance of prodromata is gradual and progressive. In certain cases the paralysis is an early symptom, in others the defects of articulation and deglutition are more prominent; in other cases psychical disturbances are decided, while in still others coma or convulsions are the striking features. The predominance of these different symptoms depends very much upon the region which suffers the most from the violence of inflammatory action. It must be borne in mind that the disorder is, as a rule, attended from the first by febrile disturbances, and that all the symptoms are those indicative of a hyperæsthetic state of the cerebrum. Should the patient survive the immediate violence of the attack, he may recover to some degree. The temperature and pulse are lowered; the active evidence of the central disease subsides, but it is not common for any amelioration of the paralysis to take place. The headache may become

more localized and less intense, or may subside altogether, and it may only reappear when the patient is fatigued. He may remain in this condition for several years. In one case that came under my observation I accidentally found a large abscess about the size of a horse chestnut in the white matter of the anterior lobe of the right hemisphere. The individual had died of phthisis, and during life complained of no symptoms which would direct suspicion to the brain lesion. He had had a febrile attack six years before, which was probably the time at which the abscess was formed. In many cases cerebral abscess follows disease of the temporal bone, and in the majority of instances it is not essentially necessary that there should be complicating general meningitis, though such is often the case.

**Causes.**—Exposure to the sun's rays, alcoholism, inflammatory disease of the bones of the head or face, meningitis, brain tumors, traumatism, and syphilis, as well as several of the zymotic fevers and rheumatism, are all predisposing and exciting causes of cerebritis. The simple form may be idiopathic, but that which results in the production of abscesses is more often due to traumatism, caries of adjacent bones, or syphilis. Jaccoud has found that the proportion of patients in regard to sex was in favor of the males, nine men being affected to every four women, and that the disease was developed between puberty and the forty-fifth year. Cerebral abscess or traumatic cerebritis may be produced, of course, at any age by injuries or the extension of other diseases. I have seen one case in which cerebritis followed otitis in a child ten years old. Lead poisoning should not be forgotten as a rare cause.

**Morbid Anatomy and Pathology.**—Cerebritis may either involve the cortex cerebri or some central parts, such as the corpora striata or optic thalami, or more rarely may affect the entire brain, but it prefers the gray matter, which is so richly supplied by blood vessels. The brain may be found to be the seat of many softened parts, and collections of pus, serous exudation from the vessels infiltrating the surrounding brain-tissue, or there may be ruptured vessels, and an escape of their contents. The brain-tissue may be stained by the hematin, and occasionally presents the appearance of simple non-inflammatory softening. The microscope enables us to see a multiplicity of changes—granular degeneration, leucocytes, broken-down nerve-elements, rarely neuroglia-thickening, and still more rarely amyloid bodies. I know of no more interesting field for the study of morbid microscopical anatomy than a brain of this kind, for nearly every appearance or grade of diseased structure may be found. The vascular lesions are capillary hemorrhage, miliary aneurism, etc. Suppuration takes place in several ways. The brain-substance may be generally infiltrated, so that it presents a yellow color throughout its extent, or there may be a localized infiltration or an encysted collection of pus. About the latter will be found a sclerosis of the brain-tissue, and about this a serous infiltration. Jaccoud has found that abscesses are more often to be observed in the white substance, in which

conclusion he is supported by the observations of many writers. Lebert,<sup>1</sup> in fifty-eight cases, found the abscess to be located twenty-three times in the left hemisphere, eighteen in the right, twice in the corpora striata, twelve times in the cerebellum, twice in the pituitary body, and once in the spinal cord. I have already presented cases which will enable the reader to appreciate the origin and size of such collections of purulent matter, and the evidences of diseased bone, fracture, etc., that are to be discerned in cases of traumatism or disease. In certain pyæmic conditions, such as erysipelas, abscesses may be found in other parts of the body as well, notably in the liver and lungs. In rare forms a rapid necrobiosis or "death" of tissues takes place, which is almost analogous with gangrene in other parts of the body, and large masses of brain-tissue are destroyed very rapidly.

Of fifteen cases of cerebral softening of acute form, Calmeil<sup>2</sup> found in one fibrine in the sinuses of the dura mater; in one, this membrane was bathed in purulent liquid, and it was also perforated at one point; in five there were recent spots of encephalitis on the right and left sides, in six on the left only, in three on the right only; in three there were cellular cicatrices in the right lobe of the brain, in one in the left lobe; in two the right hemisphere of the cerebellum was the seat of an acute inflammatory spot; in four the principal recent inflammatory spots were still in a state of red hepatization; in seven they were in a state of softening, with disintegration of the nervous substance; in four they were in a state of disintegration of the nervous substance, with a mixture of a liquid that resembled pus; in four the spots of acute local encephalitis without clot were studied microscopically. Of these, in one they were still in the state of red hepatization; the diseased regions were reddened by the widening of the capillaries, and by the presence of extravasated globules of blood: the cerebral fibres were not yet disintegrated; already small granular cells had begun to be formed in the inflamed parts. In three the nervous substance of the diseased seats was disintegrated, and more or less reduced to fragments; it was soaked in plasma, mixed with a considerable number of great cells collected together, and molecular granules; sometimes in the preparation there were seen rare globules of pus scattered. The vessels and their principal branches were constantly very apparent.

**Diagnosis.**—Cerebral hemorrhage, meningitis, cerebral tumor, embolism, and thrombosis are all conditions from which it is proper we should distinguish acute cerebritis and cerebral abscess.

Some of the symptoms of general paralysis of the insane may possibly mislead the observer. From cerebral hemorrhage we are to distinguish cerebritis by the rapid amendment of symptoms in the former, while in the latter there is progressive evidence of advancing structural changes. Fever is not connected with cerebral hemorrhage, unless there be second-

<sup>1</sup> Virchow's Archiv, x. 1866.

<sup>2</sup> Quoted by Fox.



ary inflammation of the brain-substance. The headache is not suggestive of cerebral hemorrhage, nor is the delirium or vomiting; and, after all, the only symptom which deserves attention is the paralysis. It is important to bear in mind that rigidity and contracture take place before paralysis, while we know that the converse is the rule in cerebral hemorrhage. Should hemiplegia follow a number of the other symptoms, we may consider that the hemorrhage is secondary to the cerebritis, and that some vessel has been cut across. It is almost impossible to distinguish uncomplicated cerebritis from meningo-cerebritis. The pain is perhaps more marked in the latter, and the convulsions are bilateral, and apt to be local, and due to involvement of one or more of the psychomotor centres. In uncomplicated cerebritis there is not nearly so much fever as in the meningeal form or in simple meningitis. Typhoid fever may simulate cerebritis, and *vice versa*. Attacks of the latter begin with headache, vertigo, movements of the eyes, insomnia, delirium, nose-bleed, and diarrhoea, with high evening temperature. The absence of tympanites, and gurgling in the left iliac fossa, and the appearance of paralysis and visual disorders, are quite sufficient landmarks to prevent the diagnostician from losing his way. When there is suspicion of otitis or traumatism, it is exceedingly difficult to make a diagnosis from thrombosis of the cerebral sinuses, and it is fortunate that no value is to be attached to such a diagnosis, as far as therapeutical indications are concerned.

**Prognosis.**—There is very little hope for the patient, and should he survive the acute attack he is usually left paralytic and demented. If there be a purulent accumulation, which becomes encysted, the chances of recovery are very little better, and it only becomes a question of time when the patient will die. If there be such a cerebral abscess, subsequent symptoms very much like those connected with other brain tumors will be probably developed; but, in numerous cases cited by various authors, a cerebral abscess has existed unsuspected for years.

**Treatment.**—Acute cerebritis in either form must be met with abstraction of blood, cold effusions to the head, agents which lower vascular tension, counter-irritants, and mercury in some one of its forms. The ice-bag, or the apparatus already alluded to for the application of cold water, may be used, and leeches are to be applied to the arms or behind the ears. Jaccoud and most of the clinical teachers recommend purgation, which may be obtained by the use of the compound jalap powder, followed by calomel carried almost to the point of salivation. This seems to me to be rather energetic treatment; and I think that the purgative alone, with just sufficient calomel afterward to insure moderate cathartic action, is preferable. For the purpose of diminishing vascular tension, either tartar emetic, aconite, or veratrum viride may be used. Should the cerebritis be found to depend upon syphilis or lead, the iodide of potassium may be employed as the most serviceable remedy. Blood-letting is admissible in serious cases, and is recommended by nearly all of the older writers. The head may be shaved and blistered, or cauterized; but I am convinced that sub-occipital vesication is in every way as good, and

the infliction of this punishment incident to general cauterization of the head is not warranted. Some German writers recommend the application to the shaven scalp of tartar-emetic ointment, or croton oil, and claim good results. If there be any otitis, it is well to promote otorrhœa; or, if there be a collection of pus beneath a depressed and fractured bone, it may be liberated by a free incision.

#### CHRONIC SOFTENING.

**Definition.**—A disease of the brain of a very serious character, generally of a secondary nature, and dependent upon impaired nutrition of the brain-substance through occlusion of the cerebral vessels, and symptomatized by a numerous variety of mental, sensorial, and motorial symptoms, such as mania or melancholia and subsequent dementia, headache, and cutaneous hyperæsthesia and paralysis and convulsions.

**Symptoms.**—Much confusion has resulted from the use of a variety of terms, such as “red softening,” “white softening,” “inflammation of the brain,” and other names which tend to mislead the student. For our purpose it will do to consider white and red softening as different stages of the same condition, which may result from a variety of causes; and inflammation of the brain more as the condition which I have just described than that of which I propose to speak, viz., the variety spoken of by Reynolds and others as “non-inflammatory softening.” The symptoms of softening of the brain may follow a cerebral hemorrhage, embolism, or thrombosis, or perhaps be connected with symptoms of cerebral tumor; or, again, cerebritis may leave behind it a chronic condition expressed by the symptoms I am about to detail. The early troubles of the primary form are those of intelligence; the patient loses his memory of events which have recently transpired, is unable to concentrate his attention, becomes silly, restless and irritable, quarrelling with his immediate friends, and usually getting quite excited towards night. His speech may become affected, and he sits by himself for hours during the day, and mutters constantly a mass of disconnected rubbish. This condition of stupidity increases; he may become drowsy and complain of headache, with feelings of head-pressure; he may tell us that his limbs feel heavy, and complains of muscular pain, from which he suffers in the attempt to make any movement. As to other sensory disturbances, hyperæsthesia is much more common than anæsthesia; though cutaneous areas in which sensation is impaired, are by no means rare. Motorial troubles are of later appearance, commencing with gradual loss of power of an irregular character, which affects either the arms or legs in the beginning, but finally becomes general. This paralysis is not always constant, there being a greater loss of power at times than at others. The first indication of the motorial trouble may appear either in the execution of some ordinary act, which will be performed very clumsily; or it may be shown in locomotion, when the patient will stumble or fall to the ground, as there may be a sudden giving way at the knee. When he walks he scarcely lifts his feet from the ground, but drags them after him in a helpless

manner. With the paralysis there may be a certain amount of rigidity, or tonic spasms, affecting the muscles, so that there are occasionally spastic contractions, which last for some little time. Epileptiform convulsions often occur during the disease, as well as attacks of mania, which are quite violent. When the softening is secondary, and follows an attack of embolism, thrombosis, or cerebral hemorrhage, the initial symptoms make their appearance in from one to two weeks after the occurrence of the hemiplegia. The troubles of intelligence are those which first attract our attention, and are generally connected with high temperature and severe headache. The patient may become delirious; he indulges in delusions, and grows abnormally sensitive; or, on the other hand, he is drowsy, stupid, and melancholic; and after this may follow paralytic contractures, fibrillary contractions, clonic spasms, convulsions resembling epilepsy; and he may finally fall into a state of coma. It is not uncommon for him to involuntarily pass his feces and urine. With the formation of cysts or abscesses, which constitute a late result of cerebral softening, convulsions of an epileptoid character may make their appearance; or, should the condition be acute, and result from otitis, as is the case in cerebritis, these as well as other symptoms, may be among the first to develop. Affections of speech are quite symptomatic of softening, because in so many of the cases the middle cerebral artery is that obstructed or destroyed. The hemiplegia, which may occur, is unattended by any loss of consciousness, and electro-muscular contractility is generally perfect or even exaggerated.

The following may be presented as an illustrative case:—

J. A., aged 45. The patient was brought to me by his wife during the summer of 1872. Four years before, while actively engaged in business which demanded the most devoted attention, and required a great deal of intellectual labor, he began to suffer from headaches limited to the frontal region. These were so severe that while engaged in his office he was obliged to bind a wet towel about his head. He suffered very greatly from insomnia, and found it impossible to sleep unless he took large doses of opium. He very often awoke in the night, and went upon the house-top or out into the street, wandering about the city until morning. He became very moody, treated his wife with indifference, and scolded his children without cause. He could not talk for five minutes at a time without rising and pacing furiously about the room, while he seemed to be annoyed by the slightest noises about the house. The trickling of water from the pipe over the water-closet tank, which was next to his bed-room, so annoyed him that, in a fit of impatience and ungovernable irritability, he wanted to send for the plumber in the middle of the night. His wife persuaded him to consult a homœopathic physician, by whom he was treated for nearly a year, and at the end of that time went abroad. He had meanwhile grown much worse, his mental state was much more aggravated, and his headaches, though not so severe, were still constantly present. He complained of formication of the soles of the feet, and his gait was markedly affected, both feet being scarcely lifted from the ground and he dragged one after the other when he walked. He lost rapidly in flesh, and though the sea-voyage did him



some good, he relapsed into his previous state after he reached Europe. While in Switzerland he had an epileptiform attack, and after recovery found that his right side was paralyzed. His speech was affected, and from what I can learn he must have been aphasic. The paralysis improved in a short time, and, strange to say, his mental condition also underwent a change for the better. After a few months he returned to New York, when I saw him.

He was then in an almost helpless condition, and needed the assistance of a cane and his nurse's arm to make any progress. He was bent over, and his chin was depressed, so that it almost touched his chest. The mouth was open, and the lower lip drooped slightly; while from the corners of the mouth there was an escape of saliva which trickled down over his chin. His face bore a very vacant look, and when he attempted to speak it was clouded by an anxious and discontented expression, which arose probably from the vexation he felt at being unable to speak. Phonation was not affected, but word formation seemed entirely lost, so that his attempts to speak consisted in the production of disorderly noises, the tongue being used extensively, the lips not participating. He could not protrude his tongue when told to do so. His right pupil was larger than the left. His right side was partially hemiplegic, and his wife stated that the loss of power was greater at times than at others. The right fore-arm was slightly flexed upon the arm, and the fingers seemed rigid. His control over the bladder was partially lost, and very often he would void his urine while upon the street, or at night. There is a history of trembling which affects the right arm and leg. This occurs during quiescence, and seems to have no connection with voluntary movements. His appetite is voracious, but there appears to be some difficulty in swallowing, so that it is found necessary to cut up his food. About two weeks ago he had a slight epileptoid attack. During warm days he seems disposed to sleep a great deal; but when excited by the presence of disagreeable people, or thwarted or crossed, he becomes extremely violent, and even dangerous. I saw him but once, and he was afterwards sent to an asylum.

An extremely interesting form of cerebral disease of this character, is that occurring in syphilitic subjects, and attended by narrowing of the vessels, with inflammation of their inner coats, the so-called syphilitic endoarteritis. There is consequent diminution in nourishment of large tracts of brain substance, extensive anæmia and softening.

The clinical features of such changes are numerous. In some cases the symptoms of non-specific thrombosis are presented, but the hemiplegia is rarely preceded by unconsciousness. Epileptiform attacks, severe nocturnal headache, and impairments of the mental powers are conspicuous, while a very suspicious indication of the specific nature of the trouble is local paralysis of the cranial nerves. The symptoms develop sometimes very quickly, and may disappear with great rapidity under anti-syphilitic treatment, or on the contrary, if there be much mental enfeeblement, I have found the prognosis to be grave in the extreme.

<sup>1</sup> Chauvet, <sup>2</sup> Fournier, and <sup>3</sup> Mickle, and others have described a

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<sup>1</sup> Influence de la Syphilis sur les M. du S. N., 1880.

<sup>2</sup> La Syphilis du Cerveau, 1879.

<sup>3</sup> Br and Foreign Med Chir. Review, April, 1877.

spurious form of general paralysis, which is, in reality, a form of cerebral softening. It is the same disease as that denominated by Voisin—*l'encephalopathie syphilitique*. In this pseudo-general paresis there is hebetude, delirium and incoherence. Unlike true general paresis, however, the insane delusions do not possess the extravagance of the latter, and there is very little of the boasting and inordinate vanity.

The disorders of motility are not so conspicuous as in the well recognised disease of non-specific origin, for there is not so much tremor. Labial tremor, according to Mickle, is much less common and violent, and, he says, that where such tremor exists it is always preceded by paralytic troubles, which is not the case in the ordinary paresis. An attack of hemiplegia is, as a rule, the first indication in the syphilitic subject, and the patient presents the peculiar cachectic appearance. A symptom referred to in another part of this work, and one which is pathognomonic, I believe, is the peculiar asthenic character of the mental trouble. There is a true enfeeblement of the intellect, which in some respects, resembles dementia. Memory, in regard to remote events, appears to be blunted, as well as in regard to events that have occurred recently. There is not, of necessity, much emotional irritability upon the part of the patient, although early in the trouble there is sometimes cerebral irritation and mental excitement. A disposition to sleep is not rare, and such sleep is usually quiet and may even approach stupor. In cases of syphilitic cerebral disease of every kind, the careful practitioner should be on the lookout for tertiary skin lesions and evidences of early general symptoms. In cases I have treated from time to time there has been severe neuralgia, which was much more intense at night than during the daytime, and besides, the facial and sub-occipital pain there has been a sense of vertical head pressure. The localized paralysis may involve organs which, as a rule, escape involvement in organic disease. In three of my cases there has been aphonia as a result of paralysis of the vocal cords, and in one of these cases there was, in addition, paralysis of the third nerve, and in another, alternating hemiplegia.

**Causes.**—First and foremost are primary forms of disease, which either produce occlusion of an artery, or irritation from a blood-clot or tumor. Vascular degeneration, which may result from general disease, or renal trouble, acts as a predisposing cause in the development of cerebral softening. Intellectual fatigue, sexual excitement, alcoholic intoxication, head injuries, and local disease act as exciting causes. Exposure to cold has been given as a cause of cerebral softening, and exposure to the direct rays of the sun may induce the condition. Bamberger<sup>1</sup> has observed it as a consequence of typhus and acute articular rheumatism; and Jaccoud<sup>2</sup> considers that it may be produced by syphilis in two different ways, either by a gummy tumor, which gives rise to irritation of

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<sup>1</sup> Würtzburg Verhandlungen, 1856.

<sup>2</sup> Pathologie Interne, tom. i. p. 177.

the tissue in the neighborhood, or by infiltration. According to Fournier and Huebner, syphilitic cerebral trouble may begin as late as the twentieth year of the disease, and according to the latter, as early as the first year, though it is usually until three or four years after the primary sore.

Cerebral softening is more common among people of an advanced life as an idiopathic affection, and unless it follows embolism, injuries, or like causes, is quite rare in early life, Andral having found only 39 cases out of 153 in persons under 40. Durand-Fardel<sup>1</sup> presents the following statistics regarding the period of life at which the softening began:—

|      |    |    |              |    |
|------|----|----|--------------|----|
| From | 30 | to | 40 . . . . . | 3  |
| "    | 40 | "  | 50 . . . . . | 8  |
| "    | 50 | "  | 55 . . . . . | 2  |
| "    | 60 | "  | 65 . . . . . | 5  |
| "    | 66 | "  | 70 . . . . . | 9  |
| "    | 71 | "  | 75 . . . . . | 13 |
| "    | 76 | "  | 80 . . . . . | 10 |
| "    | 80 | "  | 87 . . . . . | 5  |

Jaccoud is of the opinion, which others hold, that males are more commonly affected than females. Season has nothing to do with its development.

**Morbid Anatomy and Pathology.**—There has been great difference of opinion in regard to the pathology of brain softening. Those who described it in the early part of the century considered it to be an inflammatory affection, while Rostan,<sup>2</sup> who reported many cases, recognized a non-inflammatory form which he had met with among old people with rigid arteries. As Russell Reynolds<sup>3</sup> very properly observes, "much confusion has arisen from a tendency to misinterpret morbid anatomical appearances, without paying sufficient attention to their mode of origin." Cruveilhier<sup>4</sup> considered two forms, one of which was apoplectic, or "apoplexie capillaire," which he did not consider inflammatory; and, later, Andral<sup>5</sup> announced his disbelief in the necessarily inflammatory origin of the disease, and considered it due to occluded arteries and insufficient nutrition. Among the powerful advocates of the inflammation theory are Durand-Fardel<sup>6</sup> and Gluge,<sup>7</sup> while upon the other side may be mentioned such additional names as Kirkes,<sup>8</sup> Laborde,<sup>9</sup> Hughlings Jackson,<sup>10</sup> and many others. It may be said, I think, that softening of the brain is nearly always of an inflammatory character when it follows head injury and diseases of the cranial bones, while the majority of cases, which are second-

<sup>1</sup> *Traité du Ramollissement*, etc. Paris, 1843. P. 491.

<sup>2</sup> *Récherchés sur le Ramollissement du Cerveau*, 1820.

<sup>3</sup> *System of Medicine*, vol. ii. p. 461.

<sup>4</sup> *Etude de la Med.*, etc., 1821.

<sup>5</sup> *Précis d'Anatomie Path.*, 1829.

<sup>6</sup> *Traité du Ramollissement du Cerveau*, Paris, 1843, and *Maladies des Veillards*.

<sup>7</sup> *Comptes Rendus*, 1837.

<sup>8</sup> *Op. cit.*, vol. xxxv. p. 821.

<sup>9</sup> *Le Ram. et la Cong. du Cerveau*, Paris, 1859.

<sup>10</sup> *Op. cit.*



ary to occlusion of vessels, are dependent upon general disease of a non-inflammatory nature.

If the disease be primary, Jaccoud considers that the lesion will be of the first form, that is, at a single point; but that when the softening follows typhus fever, puerperal, and other general diseases, the foyers will be multiple. If the softening results from embolism or thrombosis, or, in fact, from any other condition producing obstruction of the circulation, there will first be a congestion with exudation of serum, hyperæmia of the vessels, and perhaps capillary hemorrhage, which is attended by coloration of the parts in the neighborhood, so that they become of a bright pink or red color, and are limited by other regions, which are anæmic and blanched, and a condition which has been called "red softening" exists. If this morbid process takes place in the gray matter, the hemorrhagic spot will be of a much darker color, and much more sharply circumscribed. The next change takes place within a week or two, when the color of the lesion becomes much more pale, and the exudation granular; fatty degeneration takes place, the softened spot extends, the neuroglia-cells, nerve-fibres, and nerve-cells become disintegrated, the axis

Fig. 23.

DIAGRAMMATIC.



TISSUE CHANGES IN SOFTENING. A. Vessel. B, B, C. Nerve-tubes. D. Gluge's corpuscles. E. Swollen nerve-tube.

cylinders disappear, and the blood vessels alone may be distinguished, and even they are greatly disorganized. At this stage the softened spot becomes much paler, is creamy in consistence, and contains stringy flakes of a fibrinous nature. It is extremely rare for resolution to take place even in the earliest stage. A form of softening, alluded to by Jaccoud, Durand-Fardel, and others, consists in the formation of yellow plates, chiefly in the convolutions (plaques jaunes) which are the result of a partial metamorphosis of the softened patches. There may be also a retrograde change, as is witnessed in the formation of cysts, which are filled by a

chalky fluid containing fat globules. There is always present a variety of cells known as Gluge's globules, which are composed of collections of small granular bodies, sometimes surrounded by a cell wall, and these are produced by the degeneration of neuroglia-cells, the debris of which are aggregated as masses of fatty granules. These little bodies, which rarely exceed  $\frac{1}{500}$  of an inch in diameter, have been found by Reynolds, Turck, and Bouchard in the cord, where their form of origin is the same.

The various colors may be seen in the brain at the same time, patches of red, brown, yellow, or white denoting different stages of the morbid process. The lighter shades generally indicate advanced stages, such being the opinion of Durand-Fardel. Charcot and various observers have found forms of white softening in old people; and others, among them Cotard, Prévost, Bastian, and Reynolds have seen cases of the same kind. It is extremely doubtful whether the condition of degeneration was not preceded by some exudation of blood-elements, and, if it was not, whether the condition had not been confounded with sclerosis. Softened patches may be in the second stage removed by allowing a stream of water to fall upon the cut surface, and when the disorganized tissue is washed away a depression is left. If the cut be made through a brain which presents the appearance of red softening, the affected patch will be found to stand slightly above the normal tissue, and this is probably due to a hyperæmia of the capillaries of the part. This fulness of the capillaries is undoubtedly due to collateral circulation of blood through the vessels contiguous to that obliterated, the normal functions being increased through double duty imposed upon them. This is the view held by Weber,<sup>1</sup> as well as by Prévost and Cotard.<sup>2</sup>

If the yellow appearance of the softened patches be not due to altered coloring matter of the blood such as we find in the early stages, it may be found later in connection with gelatinous circumscribed masses scattered through the brain or about old clots or tumors.

The parts most liable to this change both in chronic and acute forms are the corpora striata, optic thalami, white substance of the hemispheres, and sometimes the cerebellum; or there may be multiple foyers scattered through different parts of the brain.

Durand-Fardel<sup>3</sup> has collected sixty-two cases of his own and from the writings of other authors, in which the locality of the softening was the following:—

|  |    |
|--|----|
| Convolutions and white substance . . . . .   | 22 |
| Convolutions alone . . . . .                 | 6  |
| White substance alone . . . . .              | 5  |
| Corpus striatum and optic thalamus . . . . . | 6  |
| Corpus striatum alone . . . . .              | 11 |
| Optic thalamus alone . . . . .               | 4  |

<sup>1</sup> Handbuch der Allgem. und Spec. Chirur., 1865.

<sup>2</sup> Gaz. Med. de Paris, May 19, 1866, p. 336.

<sup>3</sup> Op. cit. p. 2.

|  |   |
|--|---|
| Pons Varolii . . . . .                     | 3 |
| Crus cerebri . . . . .                     | 1 |
| Corpus callosum . . . . .                  | 1 |
| Walls of the ventricles (septum) . . . . . | 1 |
| Fornix . . . . .                           | 1 |
| Cerebellum . . . . .                       | 1 |

The invasion of the brain by syphilis is usually coincident with that of other organs, notably, the liver. The morbid process prefers the central arteries, but those of small size in every part of the brain may be the general seat of inflammation and narrowing, and as a consequence a large mass of nervous tissue may be deprived of its nourishment and undergo an alteration resembling that which attends non-specific softening. The irritation of the syphilitic virus undoubtedly sets up an inflammatory process beneath the endothelium of the vessel with deposit of granular substance, nuclei and spindle-shaped cells. There is thickening of the endothelium and separation of this coat from the others. Subsequent organization of the sub-endothelial deposit and division with strata. The vessel becomes surrounded by new tissue which is also more or less organized and is ultimately supplied by capillaries. The next stage is marked by closure of the vessel.

**Diagnosis.**—In an excellent lecture delivered by Hughlings Jackson,<sup>1</sup> he says: “I do not see how the diagnosis that there is actual softening of the brain is in any case to be possibly arrived at, *unless the patient has certain local paralytic symptoms, as hemiplegia, or some other symptoms implying a local cerebral lesion, such as affection of speech*; or, again, unless there be signs of cerebral tumor (severe headache, urgent vomiting, and double optic neuritis) or evidence of injury to the head. For, so far as I know, cerebral softening is always local; I know nothing of general or universal softening of the brain. To be warranted in diagnosing softening, you must have symptoms which point to local disease. I do not say that local cerebral softening cannot exist without localizing symptoms. I only say that in their absence you are not warranted in diagnosing its existence.” This remark is made in connection with the lecturer’s disbelief in various forms of functional disease which are so often improperly called “softening,” and in which a few functional symptoms which disappear under appropriate treatment are vested by the careless or unscrupulous practitioner with an importance they do not deserve. These symptoms are those which follow depraved states dependent upon venereal excesses, fright, and other causes which lower the tone of the nervous system. Jackson’s warning is a pertinent one.

If we have hemiplegia, some renal or cardiac disease, and valvular deposits, with murmurs, our suspicions of softening generally turn out to be well founded. The history of the antecedent attack, should it be thrombosis, embolism, or cerebral hemorrhage, has much to do with the making of a correct diagnosis. As I have said, hemiplegia, unattended by loss of

<sup>1</sup> London Lancet, Sept. 4, 1875.



consciousness at the outset, is a diagnostic point in favor of softening, and suggests embolism, and if the train of symptoms given on a previous page is afterwards expressed, there can be little doubt as to the nature of the disease. A point insisted upon by Jackson is that the general mental symptoms of softening are either expressed before the softening, or follow it. He denies that *general* mental symptoms (wandering, delusions, etc.) are *directly* caused by the softening, but that *special* mental symptoms (affection of speech) are. The general mental symptoms follow a few hours or days after the local softening. The "*preceding mental symptoms*" are irritability and altered disposition.

Chronic meningitis may resemble cerebral softening, but in the former the pain is more diffused, and the motorial phenomena (spasms, etc.) are more pronounced. Softening with tumor may be made out from the additional presence of optic neuritis, choked disk, and vomiting. Some forms of progressive meningitis, such as pachymeningitis with cerebral hæmatoma (*vide* the case detailed in the chapter upon pachymeningitis), may closely simulate cerebral softening, and very often the diagnosis is exceedingly difficult, or may be impossible. The symptoms of hemorrhage from rupture of a meningeal vessel, such as occurs in the course of these chronic varieties of meningitis, may closely counterfeit the apoplectic attack which occurs so often in cerebral softening.

**Prognosis.**—Cerebral softening is one of the most unfavorable conditions with which we are acquainted. Death follows the establishment of the morbid condition sooner or later in nearly all cases occurring in adult life. An occasional case of recovery may be encountered in a young subject, but this is exceptional. Of 109 cases of both forms of cerebritis collected by Aitkin,<sup>1</sup> he found that the duration of life in cases of this disease was the following, which also proves that there are more cases of the acute than the chronic form of the disease.

| 1 died in 12 hours. | 2 died in 12 days. | 1 died in 30 days. |
|---------------------|--------------------|--------------------|
| 1 " 15 "            | 3 " 13 "           | 1 " 36 "           |
| 1 " 24 "            | 3 " 15 "           | 2 " 47 "           |
| 1 " 32 "            | 1 " 16 "           | 1 " 49 "           |
| 5 " 2 days.         | 2 " 17 "           | 1 " 60 "           |
| 9 " 3 "             | 4 " 18 "           | 1 " 65 "           |
| 5 " 4 "             | 5 " 20 "           | 1 " 68 "           |
| 4 " 5 "             | 3 " 21 "           | 1 " 190 "          |
| 7 " 6 "             | 1 " 12 "           | 1 " 220 "          |
| 8 " 7 "             | 1 " 23 "           | 1 " 5 months.      |
| 8 " 8 "             | 1 " 25 "           | 2 " 6 "            |
| 3 " 9 "             | 1 " 29 "           | 1 " 1 year.        |
| 5 " 10 "            | 4 " 30 "           | 2 " 3 years.       |
| 4 " 11 "            |                    |                    |

The greater number of these patients died, it will be seen, before the twelfth day.

The experience of other observers is slightly different from this, as

<sup>1</sup> The Science and Practice of Medicine, vol. ii. p. 304.

many persons with secondary softening have been found to live for years after the commencement of the softening. These cases being all fatal, we have to remember as well that there are many instances in which an abscess forms and becomes encysted, or the non-inflammatory softening circumscribed.

In syphilitic arterial disease the prognosis is bad when the mental symptoms are at all prominent; but, light symptoms chiefly of cerebral irritation, which indicate the beginning of the morbid process described upon another page, are sufficiently suggestive to enable us to give the patient encouragement, and to expect benefit from energetic anti-syphilitic treatment.

**Treatment.**—Our efforts should be to improve, as rapidly and fully as possible, the patient's general condition. For this purpose we must not only prescribe for him a hearty hydrocarbonaceous diet, but we are to insist upon cold-bathing, out-door exercise, and moderate stimulation. As medicaments, I am positive that there is no better remedy than phosphorus, which may be given in combination with cod-liver oil, or in solution in absolute alcohol. The bromides may be given in combination with lupulin, if there be headache or delirium; or *cannabis indica*, as recommended by Reynolds. If the bowels be sluggish, a free use of saline cathartics is of great benefit; and to relieve the head symptoms, leeching may do much good. In the chronic form tonics are indicated, and for this purpose I prefer the ammonio-citrate of iron. I am not in favor of strychnine, and should hesitate to use it if the case were at all acute.

For the relief of the syphilitic form of disease we may follow the treatment insisted upon by Dr. Taylor and others—"iodine and mercury in heroic doses."

The iodide of potassium should be employed in commencing doses of fifteen grains, and, if borne well, may be increased even to one drachm three times a day. This drug should be given well diluted and after eating. Simultaneous inunction of mercurial ointment greatly helps the action of the iodide.

### ASEMASIA<sup>1</sup> (APHASIA).

**Synonyms.**—Aphemia, Alalia, Laloplegia, Paralalie, etc.

**Definition.**—We may define *aphasia* (which is derived from the Greek *a*, priv., and *φασις*, speech) as a partial or complete loss of speech, which does not depend upon any vocal or lingual impairment of func-

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<sup>1</sup> It has occurred to me that the word "aphasia," as at present used, has too restricted a meaning to express the various forms of trouble of this nature, which not only consist of speech defects, but loss of gesticulating power, singing, reading, writing, and other functions by which the individual is enabled to put himself in communication with his fellows. I would, therefore, suggest "asemasia" as a substitute for "aphasia." The word is derived from *a* and *σημαίνω*, (an inability to indicate by signs or language).

tion, but upon disease of the speech-centres, whereby the origination of forms of expression is suspended or deranged to a greater or less degree, or a kindred loss of writing or gesticulating power. Aphasia must not be confounded with aphonia, or with the condition met with in idiots or mutes. The disease we are about to consider is seated, as it is generally conceded, in the third frontal convolution, and is characterized by the disruption of the connection between the formation of ideas and their expression by the lingual apparatus; or, as Broca has expressed it: "Le mot aphasie sert aujourd'hui à désigner la perte ou la perversion de la faculté du langage; en generale c'est de cette faculté que nous permet d'établir une relation constante entre une idée et une signe, que ce signe soit un mot, un geste, ou un trace quelconque." This loss of function varies from temporary trouble, such as the substitution of an occasional wrong word, to a condition of decided intellectual abasement. It will be well, before discussing the subject further, to say a few words in regard to the history of this interesting disease. Our first information comes from very early writers, among whom were Sextus Empiricus,<sup>1</sup> who lived two hundred years before Christ, and Pliny. Trousseau (p. 253) quotes the latter: "Illness, falls, a mere fright, impair it (memory) partially, or destroy it completely. A man struck by a stone forgot the letters of the alphabet," etc. Later, Sauvage,<sup>2</sup> Cullen,<sup>3</sup> and the two Franks<sup>4</sup> wrote most exhaustively during the seventeenth and eighteenth centuries, but all of these authors devoted more attention to mutism, aphonia, and like conditions, than to aphasia. In 1840, Lordat,<sup>5</sup> who, strange to say, became aphasic himself, described the disease under the name of alalia, a term used by Jaccoud at the present day. Though Gall,<sup>6</sup> as early as 1808, localized the speech-centre above the orbits, it was not till 1825 that its pathology and morbid anatomy were clearly settled by Bouillaud,<sup>7</sup> who, working upon Gall's theory, enunciated the doctrine that "the anterior lobes of the brain are the organs for the formation and recollection of words, or the principal signs which represent our ideas."

Afterwards, Bouillaud's views were nevertheless opposed by Andral,<sup>8</sup> Cruveilhier,<sup>9</sup> and others, to whom I shall hereafter allude. Experiments made by Marcè in 1856, and by others, confirmed all that Bouillaud had stated. The next step was taken by Marc Dax<sup>10</sup> in 1836, and by his son,

<sup>1</sup> Translated work by Huart, Amsterdam, 1725, p. 93.

<sup>2</sup> Nosologia Meth., Paris, 1722, t. ii., class 6, p. 249.

<sup>3</sup> Synopsis Nosologiæ Meth., edited by Frank, 1787.

<sup>4</sup> De Curandis Hom., Mannheim et Vienna, 1792-1821.

<sup>5</sup> Analyse de la parole pour servir à la théorie du divers cas d'alalie et de paralalie, etc., Montpellier, 1843.

<sup>6</sup> Sur les Fonctions du Cerveau, Paris, 1825, t. v.

<sup>7</sup> Treatise on Encéphalitis, p. 284.

<sup>8</sup> Maladies de l'Encéphale (Clin. Méd., 1834, t. ii.).

<sup>9</sup> Sur le principe législateur de la parole (Bull. de l'Académie, 1839).

<sup>10</sup> Lésions de la moitié gauche de l'encephale coïncidant avec l'oubli des signes de la pensée. Memoir read at the Congrès Médicale de Montpellier, 1836—Gaz. Heb., April, 1865.



who confirmed his observations in 1863. It was the younger Dax who demonstrated that aphasia was connected with right-sided paralysis<sup>1</sup> Broca<sup>2</sup> next limited the spot to the second or third frontal convolution. Since then Hughlings Jackson,<sup>3</sup> Jaccoud,<sup>4</sup> Trousseau,<sup>5</sup> Dieulafoy,<sup>6</sup> Gairdner,<sup>7</sup> and many others have added much to the interest of the subject. There has been considerable discussion as to the proper name for the affection. Lordat, to whom I have already alluded, preferred "*alalia*;" and others, among them Broca, denominated the condition "*aphemia*." The word is still used by some writers; but the term "*aphasia*" has come into general use, and is generally conceded to be much more expressive and proper than any other, but it has, I think, been too indiscriminately employed.

Jaccoud, who has rather added to the confusing nomenclature, presents a table, which embodies nothing new, and, if anything, increases the indefiniteness of our knowledge of the disease. Aphasia, or more properly *asemasia*, is most protean, as it may involve the power of reading aloud, speaking, writing, and gesticulating, in part or together, in a number of curious ways. Let us then consider the phenomena which mark its existence.

*Speech*.—The vocabulary of the aphasic patient is generally of the most limited kind, and in the beginning, should the condition follow a cerebral accident of any magnitude, his power of speech is totally absent. After a while he may be able to command one or two short phrases, or such words as "yes" or "no" in reply to every question that may be asked. These words, or such as have become automatic from constant use, are employed, and it is very curious sometimes to hear the patient give utterance to some phrase which, during health, he has constantly and sometimes unconsciously made use of. In other instances several words may be joined together in an incongruous manner; for example, it was observed, in a case I detailed when speaking of cerebral thrombosis, that the patient replied "When Benny" to the question "where do you live?"<sup>8</sup>

<sup>1</sup> Sur le siège de la faculté du langage, etc. (Bull. de la Soc. Anat., 2e, Série, t. iv., 1861).

<sup>2</sup> Gaz. Heb., April 28, 1865.

<sup>3</sup> Rep. London Hospital, vol. i., 1864, p. 388.

<sup>4</sup> Gaz. Heb. July and Aug., 1864.

<sup>5</sup> Clin. Méd. de l'Hôtel Dieu, t. ii., p. 571.

<sup>6</sup> Gaz. des Hôp., June, 1865.

<sup>7</sup> Arch. de Méd., t. ii., pp. 189-314, 1869. The reader is referred to the admirable thesis of Légroux (A. Delahaye, Paris, 1875), for a more complete bibliography of the subject.

<sup>8</sup> Numerous interesting cases are reported. One described by Osborn\* is illustrative of a form which is sometimes met with. The patient comprehended written language, and expressed himself in writing, only occasionally transposing words. He could translate fluently, and was able to calculate arithmetical sums. He could not pronounce the letters "k, q, u, v, w, x, and z," and the letter "i" seemed to puzzle him. Dr. Osborn requested him to read the following sentence from the By-Laws of the College of Physicians: "It shall be in the power of the college to examine or not any

\* Forbes Winslow, *Obscure Diseases of the Mind*, p. 343.

Durand-Fardel alludes to a patient who always gave the following absurd answer: "Madame été, mon Dieu, est-il possible, bon jour, madame." Legroux<sup>1</sup> remarks in regard to these forms: "It is to be supposed in these cases that the patients speak without hearing what they say, or that their auditory receptivity is unable to reveal the imperfection of their speech." Occasionally, however, the aphasic is conscious of the absurdity of his reply; he will laugh in a silly manner, or appear annoyed or worried, for, in a majority of cases, there is perfect mental integrity, and the position of the patient is very like that of a man driving a runaway horse. It has often reminded me of a condition which I have more than once experienced myself, and which is by no means uncommon,—the confusion of the mind during nightmare. When the individual is about to awake he is semi-conscious of the unsubstantial character of the impending danger of the dream, but cannot save himself nor can he awake. During the nightmare a person may actually spring from the bed, or make some other voluntary attempt to escape. Lordat, who was aphasic, gave, after his recovery, an account of the inward sensations that he felt during his illness, and which perfectly indicate the part played by memory. He could think, he could co-ordinate a lecture, or change its arrangement in his own mind, but he was unable, although he was not paralyzed, to express his thoughts in speaking or writing. "I thought," said he, "of the Christian doxology, 'Glory be to the Father, the Son, and the Holy Ghost,' and I was not able to *recollect a single word of it*. Thoughts seemed to arise freely, but the mode of expressing them in sounds, the receptacle of these thoughts, was forgotten."<sup>2</sup> The words which are generally lost, and are the latest to be acquired, are the pronouns and substantives, while those which the individual retains the power of articulating more than any other are the interjections, such as "Oh!" "oh, dear!" "ah, yes!" It is not rare for patients to exhibit two other peculiarities; one is a substitution of other words for those intended, the second is a conjunction of incongruous syllables; for instance, a patient may say "bel-eb" for "belief," or, as in the case reported by Trousseau, "bon-tif" was substituted for "bonsoir." Some persons are able to repeat words which are first pronounced for them by another, but are unable a minute afterwards to articulate the desired word. A patient of my own, when requested to tell what it was he held in his hand, could not say. When asked if it was a paper he shook his head; an apple? another shake, and a shrug of the shoulder; a cane? a pitying smile, and a gesture of impatience; a book? a bright smile, and the immediate ar-

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licentiate previous to his admission to a fellowship, as they shall think fit." The result was as follows: "An the bi what in the temother of the tro tho todoo to majorum or that emidrate ein einkrastrai mestreit toketra to tom breidei to ra from-treido as that kekristest." It is rare, however, for a patient to accomplish as much as this. He generally becomes impatient, and gives up the attempt after half a dozen imperfect words.

<sup>1</sup> De l' Aphasie, p. 15.

<sup>2</sup> Trousseau's Lectures on Clinical Medicine, vol. ii. p. 273, last Am. edition, 1873.

tication of the word "book." "What did you say it was?" To which there was a puzzled look, an attempt to speak, and no answer. Jackson and others have alluded to striking examples of this defect. Bastian<sup>1</sup> alludes to a form in which there was transposition of the letters, the patient saying "gum" for "mug." Patients are very apt to substitute words. Thus, when one was asked if he wanted to sit down, replied: "Give me a *bottle*, I want to *rise* down." Bauduy<sup>2</sup> alludes to a case where the connection was better shown. The man asked for a "cup of *cow*!" Some aphasics, though they may be utterly unable to speak, can sing. Hughlings Jackson<sup>3</sup> alludes to two aphasics, boys, one eight and the other ten, who could sing. Bacon reported the case of an idiot boy who was aphasic, but could sing quite cleverly. These cases are very rare, but interesting examples are occasionally brought forward. Behier reports the case of a sailor who could sing the Marseillaise, using the word "tan" throughout.

*Writing.*—The aphasic individual who cannot speak is occasionally able to write, but, in my experience, I have generally found the loss of these faculties (speech and writing power) to co-exist. This variety, which has been called *agraphia* by Ogle, has been divided by him into the anemonemic and atactic varieties. We may meet with the same peculiarities which attend the form I have already alluded to, viz.: substitution of words or letters. The patient may be able to write after a copy, but this is rare. He takes his pen and begins quite confidently, but as soon as the pen touches the paper he makes a series of scrawls, which rarely bear any resemblance to the letters forming the words he is required to write.

Bourneville<sup>4</sup> relates a case: "A woman named Justine Thomas entered the hospital La Pitié December 15, 1870, and was assigned to the service of Marotte. She became hemiplegic on the right side, and had complete aphasia. On the 18th of December the hemiplegia had nearly disappeared, but the aphasia persisted. At this time she was asked to write her name, and only succeeded in producing the appearance presented in the accompanying cut (Fig. 24, A). At different times during the year specimens of her handwriting were taken, which showed progress and marked improvement, the last attempt being made in November, 1871. (Fig. 24, B.) This lost power must not be confounded with other conditions symptomatic of insanity or sclerosis and the element of paralysis, which should be taken into account if there be any suspicion of a loss of muscular power. A hemiplegic may be unable to write simply through muscular weakness and difficult muscular coördination. Of course time will enable us to see whether the inability to write is due to this cause, or is really the "agraphic" condition. Reading, singing, and the power

<sup>1</sup> Med. Chir. Rev., xliii. p. 209.

<sup>2</sup> Lancet, 1871, p. 430.

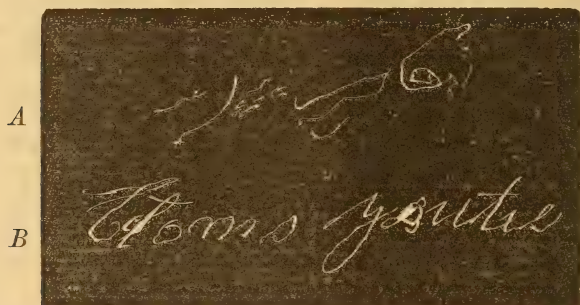
<sup>3</sup> Diseases of Nervous System, p. 412.

<sup>4</sup> Legroux's Thesis.



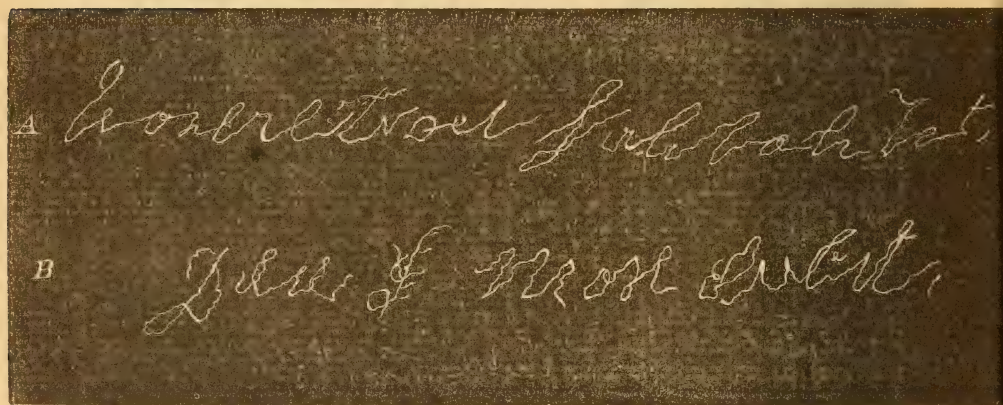
of gesticulating are lost either separately or together. A person who cannot speak is sometimes able to sing. So, too, in reading. He may

Fig. 24.



read mechanically without appreciating the sense, or may drop his words or substitute others, and perhaps is unconscious of his mistake. He may be unable to read, but may show by signs that he knows what such and

Fig. 25.



Handwriting of two patients: "A" being affected with agraphia, and "B" with cerebrospinal sclerosis. The first specimen is intended for "Possible to see you on Tuesday." The second, "Dieu et mon Droit."

such a picture may be. The power of gesticulation may be, and often is, lost. He may make attempts to describe the figure of some object, but cannot do so. Trousseau related the case of a person who was told to imitate the playing of a clarinet, but when he attempted to do so beat instead an imaginary tambourine. He is sometimes able to count figures which are before him, or pieces of money put in his hand, but if he has no such reminders, and is simply told to count, he may be able to count up to a certain number, and say ten, and does so in a peculiarly auto-

matic way. After this, when some thought is required to make combinations, the effort is unsuccessful.

For the purpose of making himself understood it is necessary that an individual should be familiar with signs (visual and auditory), which have been received either upon the retina or tympanum, and reflected upon certain ideational and receptive centres, where they are retained and serve as models for expressions the individual may wish to make in the future. The mental process which attends the formation of language or the communicating faculty becomes so intricate and *automatic* that insensibly the process of comparison and centre stimulation goes on without the knowledge of the person, and words and signs are made upon the groundwork of impressions previously received for guidance and formation. It is only when disease affects the particular centre that the harmony is lost, and the patient, though possessing the ear and eye as mentors, is unable to co-ordinate the mental factors of intelligible communication. The facility for connecting ideas with sounds or signs, which is a normal faculty, is thus spoken of by Ogle: "This faculty of converting ideas into symbols is quite distinct from that of converting symbols into ideas. The one may be acquired or lost independently of the other. Thus, a child learns to interpret the language of others before it can itself speak. Adults, as a rule, follow the same order in learning a new or foreign language. Most of us, moreover, know what it is to have the pictured map of some familiar object in our minds, yet to be perfectly unable to call up its name." This defect has hitherto been supposed to depend not upon the apparatus for the receipt of impressions, nor upon the apparatus for communication, but upon a loss of function in what has been called the "central organ of articulate speech," and that both the inability to remember words and connect them with ideas, and the inability to *compel the organ of articulation* to form words, depend upon some change only at this point. Modern physiology has taught us, however, that though the organs of reception may be healthy, there are certain cortical regions in relation therewith which seem to have a connection with the island of Reil as well, and through recent disease new perceptions cannot revive the impressions received previously in the healthy state, which have become the basis of ideas, nor can the "organ of articulate speech" be made to act, though unaffected itself. The loss of power to express ideas is symptomatized by aphasia, agraphia, or other defects in the communicating faculty. If there be *amnesia*, the central disturbance (whatever it is) is the same, and the variation of lost means for expression depends on the manner of separation of organs from mental control. There seems to be little doubt as to the seat of this centre, and as to the circumstances under which it is impaired. The collected cases of different authors mainly go to show that the left side of the brain is the seat of a lesion in its anterior part, and that the third frontal convolution is the one most constantly involved. I have already casually referred to Broca's investigations, and will now

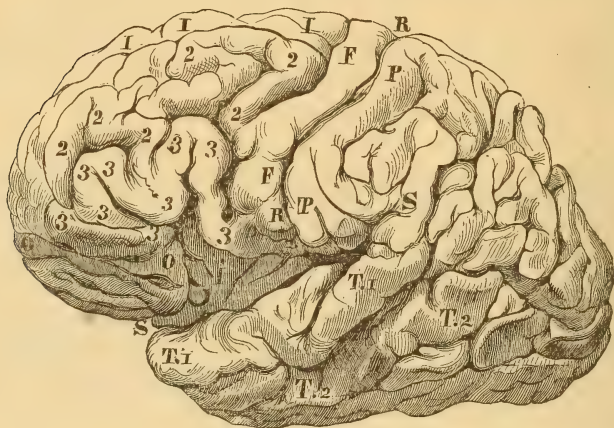
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<sup>1</sup> Journal of Mental Science.

present his description, which has been modified by Bateman,<sup>1</sup> of its anatomical seat.<sup>1</sup> “The anterior lobes of the brain comprehend all that part of the hemisphere situated above the fissure of Sylvius, which separates it from the temporo-sphenoidal lobe and in front of the furrow of Rolando (R. R.) which separates it from the parietal lobe. . . . The direction of this furrow is almost transverse; setting out from the median line, it continues almost in a direct line, and after describing some flexuosities terminates below and outside of the fissure of Sylvius, which it meets almost at a right angle behind the posterior border of the lobe of the *insula*.

“The anterior lobe of the brain is composed of two divisions, the one inferior, or orbital, formed by the several convolutions called *orbital*, which lie on the roof of the orbit, and of which I shall not have to speak; the other, superior, situated under the outer wall of the frontal bone, and under

Fig. 26.



the most anterior portion of the parietal. This superior division is composed of four fundamental convolutions called, properly speaking, the *frontal convolutions*; one is posterior, the others are anterior. The *posterior*, F F, slightly tortuous from the anterior boundary of the furrow of Rolando. It is therefore almost transverse, and ascends from without, inwards, from the fissure of Sylvius to the great median fissure, which receives the falx cerebri of the brain. This is why it (F F) is described indifferently under the name *frontal*, *posterior*, *transverse*, or *ascending* convolution. The other three convolutions of the superior division are very tortuous and very complicated, and some practice is needed to distinguish them in all their length without confounding the fundamental furrows which separate them with the secondary furrows which separate the second order folds, and which vary in different individuals according to the degree of complica-

<sup>1</sup> The reader may also consult Morel's Plate, presented upon a previous page.



tion; that is to say, according to the degree of development of the fundamental convolutions. These three fundamental convolutions, 1, 2, 3, are antero-posterior, and, running side by side, extend from before backward over the whole length of the frontal lobe. They commence on a level with the superciliary arch, whence they are reflected, to be continuous with convolutions of the inferior division, and terminate behind in the frontal transverse convolution, F, F, which all the three enter. They are called *first* (1), *second* (2), and *third* (3), *frontal convolutions*. They may also be called *internal* (1), *middle* (2), and *external* (3); but the ordinary names have prevailed. The first (1) runs along the great fissure of the brain; it presents, constantly, in the human species, an antero-posterior furrow more or less complete, which divides it into two folds of a second order; it has, therefore, been divided into two convolutions, but comparative anatomy shows that these two folds form only a single fundamental convolution. The *second* (2) frontal convolution presents nothing peculiar; not so with the *third* (3), which is more external. The latter presents a superior or internal border, adjoining the tortuous border of the middle convolution (2), and an inferior or exterior border, the relations of which differ according as they are examined before or behind. In its anterior half this border is in contact with the external border of the most external orbital convolution. In its posterior half, on the contrary, it is free and separated from the temporo-sphenoidal lobe by the fissure of Sylvius, S, S, of which it forms the superior border. It is in consequence of this latter relation that the third frontal convolution is sometimes called the *superior marginal convolution*.

“Let me add, that the inferior border of the fissure of Sylvius (S, S) is formed by the superior convolution of the temporo-sphenoidal lobe, which is therefore called the inferior marginal convolution T, T. It is an antero-posterior fold, thin, and almost rectilinear, which is separated from the temporo-sphenoidal convolution T 2, T 2, by a furrow parallel to the fissure of Sylvius. This furrow is described under the name of the *parallel fissure* (with reference to the fissure of Sylvius, S, S). Lastly, when the two marginal convolutions, *superior*, 3, 3, 3, and *inferior*, T, T, are drawn away from the fissure of Sylvius, S, S, there appears an enlarged and slightly prominent eminence, I, from the summit of which five small simple convolutions, or rather five straight folds, radiate in a fan-like manner. It is the lobe of the *insula* which covers the extra-ventricular nucleus of the corpus striatum, and which, arising from the bottom of the fissure of Sylvius, S, S, is found to be structurally continuous by its cortical layer with the deepest or most deeply seated part of the two marginal convolutions, 3, 3, 3, and T, T, and by its medullary layer with the extra-ventricular layer of the corpus striatum. The result of these structural relations is, that a lesion which propagates itself continuously from the frontal lobe to the temporo-sphenoidal lobe, or, *vice versa*, will pass almost necessarily by the lobe of the insula, and that from thence it will most probably extend to the extra-ventricular nucleus of the corpus striatum, since the proper substance of the *insula*

I, which separates the nucleus from the surface of the brain, forms only a very thin layer."

Not only may a lesion of the speech-centre itself produce aphasia, but in numerous instances (some of which have been referred to by Jackson) it may follow the destruction of adjacent parts, as a consequence of some such accident as the plugging up of the middle cerebral artery. As a consequence of such a pathological condition, a large area of brain substance will be destroyed, so that impaired mental function as well as aphasia takes place. We shall presently see that though this particular part of the third frontal convolution is the seat of the organ of "speech expression," there are other important cortical regions which, when destroyed, give rise to asemasia.

An important subject in this connection is the side of the brain which is affected. Though exceptional cases have been reported in which the right cerebral hemisphere has been the seat of the lesion, the rule is the other way. In some instances, even, no lesion whatever has been found; or, on the other hand, the left anterior convolutions have been the seat of morbid change, and no loss of speech has been occasioned. Simpson<sup>1</sup> has related one case where marked destruction of the *left* anterior lobe was observed, and yet *no aphasia existed*. This man, aged 65, who had been epileptic for ten years, having as many as three or four attacks a month, died. The white and gray matter of the left hemisphere were markedly atrophied, and there was a cavity in the left posterior frontal convolution of  $1\frac{3}{4}$  inches longitudinally, and  $1\frac{1}{2}$  transversely.

The following case is interesting, as it shows that almost complete aphasia may exist without any disease of the island of Reil:—

M. A. B., aged thirty-five years, married. Family and previous personal history good, but it is possible to trace syphilis. The patient had an apoplectic attack in August, 1859, with loss of consciousness, which lasted for two hours; on recovery it was found that she was unable to speak, but there was slight improvement after a few months. Present condition, July 17, 1874: The patient is a middle-sized woman of seemingly good condition, with the exception of her nervous trouble. There is slight paralysis of the left side; can move left arm well, but slowly, and walks with a shuffling gait. Tactile sensibility, and sensibility to differences in temperature, are decidedly impaired on the left side, on which side there is an appreciable amount of analgesia. She protrudes her tongue in a straight line, but feebly. No loss of taste or smell. Her mental condition is below the average. The first part of her history I have taken from the records of the Epileptic and Paralytic Hospital, and I also find that for some months she has been suffering from symptoms of phthisis. When I saw her on August 10, 1875, the patient was in advanced phthisis; her nervous condition was the following; Paralysis of the left side; her left hand lies in her lap, the thumb being contracted and flexed; the flexor tendons of the hand are rigidly con-

<sup>1</sup> Med. Times and Gazette, Dec. 21, 1867.

tracted, so that at the wrists they stand out like tense cords. There is very little atrophy of the left upper extremity, but there is a certain stiffness about the elbow-joints of this side. The left lower extremity seems to be nearly as strong as its fellow. Motion at the hip and knee-joints is limited. She can raise her foot from the ground when sitting, but when she walks it is in a shambling manner, dragging her left foot, or scarcely lifting it from the ground. There is some paralysis of the left side of the face, and it is impossible for her to protrude her tongue. Sensibility seems to be very slightly affected in the paralyzed side. She is almost completely aphasic, her repertoire of words being confined to "yes" and "no," the former being repeated several times in answer to any questions she may be asked. When she is asked her name, she is unable to tell it. "Is it Jane?" she shakes her head and smiles. "Is it Ann?" another shake of the head, and an attempt to speak, the only result being the production of an unintelligible noise. "Is it Mary?" when she brightens up and says, "Yes, yes, yes; Ma——" prolonged, and she generally gives it up in disgust. She cannot write, but makes a disorderly scrawl; although we learn from her friends that in health she wrote well. She gesticulates a good deal, and endeavors to attract the attention of those in the ward, and evidently appreciates everything that goes on about her. Her pupils are easily dilated, but she does not see with the right eye, and on examination I find atrophy of the optic disk. During the winter and spring of 1875-76, she seemed to suffer much from her pulmonary trouble. There was œdema of the lower extremities, which increased so that the anasarca became general, but she was somewhat relieved by digitalis and iron; diarrhœa supervened, and she finally died on the second day of June, 1876.

*Autopsy.*—The dura mater was considerably thickened, and presented the appearance of old pachymeningitis. There was no lesion to be discovered in either third frontal convolution, but an old clot was found in the right caudate nucleus. This clot was about half an inch in diameter, and was surrounded by some dense tissue. Cortical lesions were present on both sides of the brain, but of superficial extent, and confined chiefly to the parietal convolutions; these consisted of softened patches in advanced stages of degeneration. The cerebral arteries contained patches of a yellowish or atheromatous nature. The spinal cord was not examined. Both lungs were found to be tubercular, and in the middle lobe of the right there was a large cavity. I was unable to find any tubercular deposit whatever in the brain or its meninges. The left frontal convolutions were examined, but no disease whatever was found, and the occipital convolutions were in normal condition.

Hemingway reports the following interesting case of left-sided paralysis with aphasia:<sup>1</sup>

Jane R., aged 30, widow; occupation seamstress; education fair, can read and write. Entered hospital October 30, 1873. Family history good; says she always was a healthy woman till present illness. Admits having had a sore on genitals five years ago. Cicatrices are at present visible on forehead, which are probably a result of tubercular syphilides;

<sup>1</sup> Medical Record, March 4, 1876.



says they came there five years ago. Her left eye shows the result of an old ophthalmia, which, it was supposed, was of gonorrhœal origin. For two years past has had slight palpitations on exertion. Always used her right hand in her occupation. Four months ago, one night when she was going to bed she became suddenly speechless; there was no paralysis whatever. Next morning, on attempting to arise, found her left arm, leg, and side of face paralyzed; also, with loss of sensation in those parts. Loss of speech was complete; and hearing, which before this was excellent, was now lost in left ear. Her tongue was only affected in sensation; she was not able to appreciate sweet substances placed on the tongue; sense of smell also lost. About one month after this attack, *i. e.*, three months ago, improvement began in speech, face, and lower extremity, and has continued since then. Upper extremity began to improve one month ago. Sphincters have not been affected. Is a medium-sized woman, pretty well nourished; mental faculties good, with exception of loss of memory, constituting well-marked amnesic aphasia. Is unable to recollect many words, names of objects, as *hat, key, handkerchief, pencil*, etc.; though she can readily repeat them on being told, she forgets them immediately afterwards. Is unable to read continuously, omitting words, and giving up from inability to fix attention. On attempting to write the letters of the alphabet, the result was A B C D S G H I; but when the letters were separately told her, she wrote them down easily. Partial paralysis remains on left side of face; cannot close eyelids tightly. Sensation is lost to a great extent on left side of face, and in left nostril. Does not wince on the application of aqua ammonia to left nostril, nor when the conjunctiva on same side is touched with an irritant. Hearing poor on left side. Taste is impaired anteriorly and posteriorly on left side of tongue.

Dynamometer, { left, 32, } outer circle.  
right, 80, }

Æsthesiometer is valueless, on account of loss of sensation, of reaction to pain. Does not wince on pinching arm, but does on palm of hand and tips of fingers. Perception delayed; takes about three seconds. Can raise arm to level of shoulder, a little stiffly. Can flex and extend forearm and fingers, but slowly. Heart sounds normal. Walks without elasticity. Sensation in leg as in arm. Reflex action lessened. Electromuscular contractility good.

The accumulation of reported cases, however, in which the lesion was on the left side, leaves no doubt in regard to this question. Jackson and Ramskill report 40 cases of right hemiplegia with aphasia, and but one of left hemiplegia. Ogle<sup>1</sup> reports 25 cases all with the lesion in the left hemisphere, though there were morbid changes in some of these in other parts. In not one of these where the lesion was on the left side was there undisturbed speech. Magnan<sup>2</sup> reported thirty-one cases of aphasia, and in all but four was there right-sided hemiplegia. Trousseau, in 1868, had collected all the cases he could find, the number being over one hundred, and in all but ten there was right-sided paralysis. Seguin<sup>3</sup> has

<sup>1</sup> St. Geo. Hosp. Reports, vol. ii.

<sup>2</sup> Bull. de l'Académie de Médecine.

<sup>3</sup> Quarterly Journal of Psychological Medicine, 1861, xxx., 663.

collected 46 cases from the records of the New York Hospital, and in all but three there was right hemiplegia. Thus it is settled, I think, that the left side of the brain is that which contains the speech-centre.

The question as to the relative frequency of right and left hemiplegia naturally arises, and from the inspection of a large number of cases it will be seen that there is a very slight preponderance of the former.

Browne,<sup>1</sup> from Baillarger's tables, says that "in aphasia right is to left hemiplegia as 15 is to 1."

By the following table it will be seen that there is very slight preponderance of right-sided paralysis, and the comparison between the infrequency of aphasia with left hemiplegia, and the slight difference between the relative frequency of occurrence of both forms, is inconsiderable.

|                      | Cases of hemiplegia. | R.        | L.        |
|----------------------|----------------------|-----------|-----------|
| Ogle . . . . .       | 75                   | 43        | 32        |
| Andral . . . . .     | 136                  | 73        | 63        |
| Baillarger . . . . . | 110                  | 58        | 52        |
|                      | <hr/> 321            | <hr/> 174 | <hr/> 147 |

As to the exact site, Seguin tabulates 545 cases, in all of which but 31 the lesion was in the left anterior lobe. Why the left side is the seat, especially when embolism or thrombosis is the cause, has already been explained by the fact that the left middle cerebral artery is that which is in the most direct line from the heart. The next link in the chain, which is the question of valvular disease, and its connection with loss of speech, has been discovered by H. Jackson, who has found that valvular disease is nearly always associated with the hemiplegia, that is, connected with loss of speech. He has seen more than 50 of these cases.

The records of cases of right hemiplegia with aphasia in which I made autopsies, show that there were other lesions, but always some trouble in the course of the middle cerebral artery. I therefore agree fully with the majority of observers, that loss of speech depends, except in rare instances, upon lesions in the left hemisphere, but that it *may also* follow a lesion in the other hemisphere. Both Brown-Séquard and Van der Kolk have advanced theories—the first, that articulate speech is a reflex process; and the latter, that it is seated in the olivary bodies. This last view was held by Willis, Solly, and others. Laycock is of opinion that these organs are "subservient to the emotions through the muscles of the face and tongue by language, and emotional cries and sounds." And he says: "It is by no means improbable, however, that the emotional movements of the hands, as well as of the tongue and face, are likewise under their direction. They are, therefore, to be considered as regulative ganglia to the motor centres of the facial, hypoglossal, and limb nerves in the medulla oblongata belonging to the substrata of the sensory tract."

Dr. Herbert Major,<sup>2</sup> in a very complete article upon the micro-

<sup>1</sup> W. Riding Reports, vol. ii., p. 284.

<sup>2</sup> West Riding Reports, vol. vi. 1.

scopical anatomy of the island of Reil, sums up his conclusions as follows :—

“1. The cortical layers of the insula agree in number, order, and general arrangement with those of the vertex, but the cells of the *third* layer are in the insula generally smaller than at the vertex. The vessels and neuroglia present no peculiarity.

“2. The various gyri forming the insula present a similar structure.

“3. No difference of structure can be detected in the right as compared with the left insula.

“4. The method of union of the white matter with the cortex is in the insula similar to that observed in other lobes.”

The departure from the healthy state is seen in enlarged vessels, a shrunken appearance of the cells of the first layer and a diminution in their number, together with even a change in the cell-contents, the nuclei being broken down and agglomerated at the centre. The cells of the second and third layers have lost their processes, and the protoplasm contains granular debris, while the other cells of the lowermost layers suffer the same changes as well as transposition.

Aphasia may be dependent upon any form of brain disease which produces disorganization of, or pressure upon, the third frontal convolution or parts immediately adjacent or of certain cortical centers behind the fissure of Rolando.<sup>1</sup> Among the common diseases which lead to the structural changes are cerebral hemorrhage, thrombosis or embolism, tumor, or sclerosis, as well as certain forms of meningitis. Age appears to play but a small part in the production of this condition, except so far as it influences cerebral hemorrhage, embolism, or the other diseases just mentioned.

Since the appearance of the first edition of this book the study of aphasia has received fresh impetus as a result of the development of our knowledge of cerebral localization. The observations of Munk<sup>2</sup> have materially altered the views of physiologists, and the recent writings of Kussmaul, Bastian, Broadbent and others, have established the existence of the cerebral cortical centers, which play a part in *asemasia* and one of great importance.

Bastian<sup>3</sup> has formulated the ideas of modern writers as follows :—

I. Defects of verbal memory; that is defects in the association of ideal things or of conceptions with ideal words.

<sup>1</sup> Among fifteen cases reported by Sander\* there were two in which the original lesion was found in the left parietal lobe, in some of the bundles of fibres radiating from the corpus striatum.

<sup>2</sup> Ueber die Functionen der Gehirnsrinde.

<sup>3</sup> The Brain as an organ of Mind, 1880.



## A. AMNESIA VERBALE.

(a. Paralytic variety; b. Incoördinate variety.)

1. Diminished Excitability of the Auditory Word-Centres.
  2. Defective Action of the Visual Word-Centres.
  3. Damage to Visual Word-Centres and of Afferent Fibers to Auditory Centres; together with certain defects producing Incoördinate Amnesia.
  4. Damage to commissures between Auditory and Visual Word-Centres.
- II. Defects in the association of Ideal Words with verbal movements for speech and writing, or for either of them singly.

## B. APHASIA.

5. Damage to first parts of outgoing tracts leading from Cerebral Word-Centres to left Corpus Striatum.

## C. AGRAPHIA.

6. Damage to first parts of outgoing tracts leading from the left Visual Word-Centre.

## D. APHEMIA.

7. Damage (a) to first parts of outgoing tract leading from the left Auditory Word-centre, or (b) to some lower part of the same tract, or (c) to the actual Motor Centres for articulation.

The involvement of the visual and auditory centres as has been stated, even though there may be no disease of the island of Reil, accounts for the production of the various forms of *asemasia*. In well reported recent cases the matter has been definitely settled that destruction of one or both of these centres, may be followed by disruption of the corrective influence of visual or auditory associations. The individual may be unable to speak or write upon dictation, or he may be equally powerless to copy a printed page or correctly count a given number of objects or figures. The case of the late Dr. Allin, of New York, which has been ably reported by Dr. A. B. Ball, is one of the most valuable contributions to the modern literature of aphasia. In Dr. Allin's case the lesion was confined to the "whole of the inferior parietal lobule and the first temporal gyri," and no change in the Island of Reil was found. Dr. Allin visited my office some months before his death, and at various times had been seen by Drs. Ball, Seguin and Metcalfe.

The feature of his *asemasia*, was his inability to use common nouns and proper names. He was able usually to closely approach in sound the word he desired to use, but if he saw the initial or heard the first syllable, he was able to finish the rest. *At first he was unable to repeat the word after it had been pronounced by another person*, but subsequently learned a large number of words used in ordinary conversation. He was *agraphic*, and could not write at dictation though he recognized the number of letters and made them by straight lines. He was utterly unable to comprehend auditory symbols. Dr. Ball said to him, "Dr. Peters called to see you;" he replied, "I don't know him." The name was repeated to

him several times, but he failed to recognize it, although it was the name of an intimate friend. The written name was then shown him; "What a fool I am," he exclaimed, "of course I know him." He afterwards said, "The words I can't pronounce are the words I can't hear." This indicated a disruption of the auditory control. Dr. Allin could sing, gesticulate, but manifested a peculiar symptom—the reversal of the position of objects he took up. For example, he placed his knife and fork with their points toward him upon the table, but he immediately recognized his mistake. When he read aloud he seemed to have lost the guiding control of the ear, for he often used the wrong word.

The failure of the visual and auditory centres gives rise to many interesting phases of hindered speech action and writing power. Bastian illustrates the table I have just given by several groups of cases of which the following are examples:—

1. An individual who could articulate distinctly any words that occurred to him either spontaneously or when they were pronounced slowly and loudly, but could not speak at other times. He could read aloud from printed copy, but could not repeat the words he had seen the moment before. Here was a case in which the auditory centre was needed and when words were not properly revived by volitional excitation.

2. An individual who could repeat spoken words but could not read aloud.

3. Dr. Banks' case of the man who had lost the power of apprehending what was spoken by others with loss of comprehension of written or printed characters.

The explanation of word blindness recently given by Magnan<sup>1</sup> bears out the investigation of Ferrier and Tamburini. Magnan considers that there are two centers which are involved; the visual perception goes first to the corpora geniculata or some other center in their neighborhood (see Charcot's plate,) and from them to the angular gyrus, where it is made the basis of psychic action involving an exercise of memory and reasoning power. The disruption of this center with the island of Reil, eventuates in the phenomenon of word blindness, as the idea cannot form expression. Two cases have been presented by him.

Very few examples of aphasia in very young persons have been reported, for vascular neuroses are quite unusual among children, and right hemiplegia, with a lesion in this particular part of the brain, is of rare occurrence. A case was reported by Eulenburg which was quite unique.<sup>2</sup> The patient was eight years old; two years before he had had scarlet fever, and six weeks after the development of the disease there were convulsions and coma, followed by right hemiplegia with aphasia. The paralysis almost subsided in two weeks. He speaks but two words, viz.: "Ach," which he always uses for "mien," and "Ja," with which he answers all other questions. The fact that dropsy and albuminuria had ex-

<sup>1</sup> Gazette des Hôpitaux, Jan. 24, 1880.

<sup>2</sup> Berlin Med. Gesellschaft, July, 1869,

isted induced the author to infer the presence of softening of the central organ of speech.

Aphasia of a temporary character may depend upon functional conditions, such as cerebral congestion, indigestion, or as the result of fright or other emotional forms of excitement, or may be connected with epilepsy or hysteria. Kisch<sup>1</sup> reports three cases of transitory aphasia due undoubtedly to cerebral congestion. One of these was a very stout woman who, having drank a very large quantity of carbonic acid water, fell to the floor after being dizzy, but did not lose consciousness. This seizure was followed by headache, and later by complete aphasia. She subsequently recovered. Two cases of aphasia of a similar character are reported by Berger.<sup>2</sup>

Habershon<sup>3</sup> presents an example of aphasia which was caused by fright. A much more rare variety of the disease is that which is connected with epilepsy. Three such cases were published by Allbutt.<sup>4</sup> One of these patients fell, striking on his left temple; some time afterwards epileptiform attacks appeared with paralysis of the right arm and leg. The second case was that of a woman aged fifty, who had had epileptic convulsions of a bilateral character for two years. After the attack she was somewhat aphasic, and "had a mental vision of the words," but was unable to speak them. This condition of affairs lasted for two hours. The third patient was a man, thirty years of age; there was no loss of consciousness, but attacks of hyperæsthesia in the right arm and hand, followed by blindness, lasted for twenty minutes or longer, and were succeeded by speechlessness lasting two hours.

**Diagnosis.**—In making the distinction between aphasia and other difficulties of speech, we are apt to be misled by defects in articulation, dependent upon inco-ordination or paralysis of the tongue, or by certain mental irregularities, or sometimes by congenital mutism.<sup>5</sup> We are to bear in mind the fact, that there may be transitory aphasia, but that organic disease of the speech-centre is generally of permanent duration; and that there are but very few exceptions to this rule. The speech defects which are of a local character are symptomatized by the patient's inability to speak at all, though he may fully convince us of his ability to form words and appreciate their meaning; and, moreover, he can always, should there

<sup>1</sup> Berliner Klin. Wochenschrift, 1869, 433.

<sup>2</sup> Wien. Med. Woch., 1869, 102.

<sup>3</sup> London Lancet, 1870, vol. ii. 402.

<sup>4</sup> Med. Times and Gazette, 1869, vol. i. p. 491.

<sup>5</sup> Dr. Browne,\* of the West Riding Asylum, recently examined 29 cases of morbid affections of language, or all in the existing population of the Crichton Institution at Dumfries; 14 of these were females, and 15 males. Of these, which he arranged in three classes, he found among the women: "1. Intermittent mutism 5, in one connected with the catamenia. 2. Constant mutism, 7: of these one had been a public singer; 1 when roused could with difficulty articulate, having facial paralysis; 1



not be paralysis of the hand or forearm, write any word that he may wish to speak. This is not the case in aphasia. In lighter forms of tongue paralysis there is no trouble about the selection of words, but simply a clumsiness in pronunciation, and in many of these forms evidences of local muscular weakness, in connection with the speaking apparatus, draw attention to the real nature of the trouble. A disease presenting these local defects is a so-called glosso-pharyngeal paralysis. The same condition of affairs is met with in general paralysis of the insane, but with this, as well as other troubles of the same kind, there are various other symptoms which accompany the speech defect, such as mental impairment, with peculiar delusions and muscular trembling. Hysteria sometimes gives rise to a very curious speech derangement, which, in its strictest sense, can hardly be called aphasia. The patient occasionally introduces obscene and profane words in place of others more conventional. A form of speech trouble described by Winslow<sup>1</sup> and Romberg<sup>2</sup> is expressed by mimicry of individuals, who speak to the patient or who talk within ear-shot. He closely imitates the tones of their voices and mannerisms, and repeats the words addressed to him, besides mimicking their gestures and attitudes. These phenomena are occasionally seen among the insane. Romberg has called this morbid state *echolalia*. I have at present a case under observation who is an example of this kind, only his infirmity does not exist to so marked a degree as in the cases of the two observers above mentioned. My patient is an idiot, and possesses but very little mental power. He can point to his mouth, places his hand upon his abdomen when hungry, and can call attention to his bodily needs by equally simple gestures, but beyond this he is more an automaton than a living being. When asked a question, for instance, "How are you?" he repeats the two last words, "Are you?" and "Why don't you answer?" he replies, "Don't you answer?" He invariably repeats the last two or three words

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could not walk in consequence of spinal deformity; 1 was an idiot laboring under phthisis; 1 uttered cries when suffering pain. 3. One was reduced to monosyllabic utterances. 4. One manifested incessantly, day and night, irresistible loquacity.

Among the males: "Intermittent mutism, 1. 2. Constant mutism, 5: in 1 the mutism is of 20 years' duration; in 1 it is accompanied by tremor of the limbs; in a third, who attempted to cut his throat, there is unintelligible muttering soliloquy. 3. One was reduced to monosyllabic utterances. 4. Two manifested constant loquacity: in one, an idiot, there is congenital left hemiplegia; in the other, who is healthy, the loquacity is so great and rapid that the words run into each other so that he seems to speak in long sentences. 5. Two present symptoms of general paralysis; the articulation is indistinct or unintelligible. 6. In one case there appeared to be the omission of the first syllable of every word, followed by alternate mutism and loquacity. 7. In one, an idiot, language is limited to a few words, and these are exclusively oaths, with congenital right hemiplegia, and club-foot. 8. Two idiots emit nothing but acute inarticulate cries; one roars like a wild beast." There was no paralysis in these cases except of the face in two general paralytics, and of the lower extremities in two idiots, the paralysis in these latter cases being congenital.

<sup>1</sup> *Obscure Diseases of the Brain and Mind*, Am. ed. p. 343.

<sup>2</sup> *A Manual of the Nervous Diseases of Man*, Syd. Trans., vol. ii. p. 431.

of any question that may be put to him, so that his answers are but echoes of the questions. Such a defect is explained by Bastian by the fact that "the auditory word centres respond only to direct 'sensory' incitations, and not at all to those of the 'associational' or 'volitional' types."

In the early speech disturbances of left hemiplegia, or organic diseases of the brain, the patient's attempts to articulate will result in a clumsy and mispronounced word; while in aphasia his articulation, be it ever so limited, is rarely imperfect, his "yes" or "no" being fairly pronounced, or, if he has improved so far as to be able to pronounce but a part of a word, he will do this distinctly, while perhaps the other syllables will either be not pronounced at all, or in such a way as to be utterly unintelligible. There are generally with the aphasic great impatience and embarrassment, mimicry, and gesticulation, which are evidences of mortification arising from the knowledge of his failing, and his gestures take the place of words. In agraphia the handwriting or results of attempts at writing must be compared with specimens, such as would be made by patients who are insane, ataxic, or paralyzed, and it is necessary for us to carefully note the omission of words, or combination of syllables which bear no relation to one another, as well as the character of the patient's composition. If he be insane, he will not admit any absurdities to which he may give expression, but with the aphasic the case is different, for he always evinces his chagrin when he finds that he has written the wrong word, and endeavors to correct his mistakes. There are cases spoken of by Bacon<sup>1</sup> and others, in which the only evidence of the patient's insanity is his writing, but even here the defect is more in the expression of a disordered mental state than in an impairment of the communicating faculty. The handwriting of the general paralytic sometimes closely resembles that of the aphasic patient, but in the first, with time there is progressive impairment, while in the other, if anything, there is improvement.

The medico-legal questions which may arise in regard to the responsibility of aphasia are worthy of consideration. The aphasic of course may suffer an intellectual impairment, which lasts a short time after the attack. This is not necessarily accompanied by a loss of judgment. It is more a condition of mental sluggishness, and it will not do to say that the individual is incompetent. The aphasic makes intelligent efforts to communicate, even though he may not be able to do so. He gesticulates, and tries to explain himself, and the expression even of his eyes tells of everything but intellectual unsoundness. Additional evidence of softening in dementia throws an entirely different light upon the matter, but even then it must be remembered that aphasia is not necessarily associated with such states.

A case of interest is reported by M. Lucas Championnières:<sup>2</sup> "The question was raised in this particular instance *a propos* of a case in which the patient, in spite of an enfeebled intelligence, had become ca-

<sup>1</sup> On the Writing of the Insane, p. 12.

<sup>2</sup> Journal de Med. et de Chir. Prat., abst. Br. Med. Journ. Sept. 15, 1877.

pable of writing with the other hand. He could not, however, write if left to himself, and could only recopy what was written and set before him, and the expert physicians vainly tried to make him recopy a power of attorney or a will, while he willingly wrote any ordinary phrase or document which did not bind him to anything. This man, then, knew perfectly what he was doing, and the Société de Médecine Légale concluded that he possessed still thorough intelligence and free will to be able to continue to enjoy his civil rights, the intellectual debility which he had suffered not appearing to be sufficient to justify what the French laws call an 'interdiction.'" The society recommended that he should be taken care of by a "council," so that he should be guaranteed protection against danger that might arise in the condition of his affairs.

We must bear in mind the existence of heart trouble should it exist, or vegetations and other indications of extraneous disease which might lead to the causation of thrombosis or embolism.

In regard to the diagnosis of aphasia it may be said upon the authority of Seguin that "predominant word deafness or word blindness, with hemianæsthesia, cutaneous, muscular or sensorial, is dependent upon a lesion placed behind the fissure of Rolando in regions which correspond with the sensory cortical centres."

**Prognosis.**—The view we are to take of our patient's condition is to be governed entirely by the question whether there is or not a primary organic disease, its importance and the character of the aphasia.<sup>2</sup> In the light forms, such as result from fright and cerebral congestion, or those connected with hysteria, the prognosis is exceedingly good, and the same is the case when it is the result of protracted fever. Legroux (op. cit. p. 60) speaks of an aphasia of quite temporary duration, which is occasionally of gouty origin, or connected with diabetes or albuminuria. <sup>2</sup> Dr. Rotch has also described varieties of temporary aphasia met with in patients who are the subjects of Bright's disease, and presents two cases. The prognosis of the condition itself is quite good, but a serious indication of grave cerebral trouble. Aphasia with paralysis is always significant of deep trouble. Such an aphasia, when it occurs with hemiplegia, may persist perhaps during the individual's lifetime, and after every vestige of the hemiplegia has disappeared. If there be softening, or previous acute cerebral disease, or if there be evidence of arterial degeneration, or valvular deposits, the case assumes a hopeless aspect, and may be nearly always pronounced incurable. Aphasia as the result of traumatism is occasionally relieved by surgical interference.

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<sup>1</sup> In one case reported by Bateman, the patient recovered almost entirely, and he could pronounce every word distinctly, with the exception of those containing the letter P.

<sup>2</sup> Boston Medical and Surgical Journal, May 26, 1881.



**Treatment.**—Our first indication is to improve, if possible, the organic disease, and sometimes we are able to better the patient's condition to a great degree. Should there be hemiplegia, contractures, or other evidences suggestive of degeneration of the cerebral tissue, we will find ourselves powerless to help our patient materially. It is only when aphasia exists as an isolated symptom that very active measures are followed by some show of success. In such a case local blood-letting, purgation, and the use of ergot, and the bromides, may completely relieve the condition; and even when the disease is established, and the destruction of the speech centre has been limited, there is a possibility of improving the patient's partially lost faculty. Systematic education, and the training of the left hand, and the *development of the right side of the brain*, may result in an increase in the patient's faculty of communicating. In rare cases, viz., those of traumatic origin, it may do well to use the trephine. Broca, under the heading, "*La Topographie Cranio-Cérébrale*,"<sup>1</sup> described experiments made by him to determine the relation of the cranial bones with underlying parts; and Turner<sup>2</sup> has made additional observations, and given rules for determining this relation.

## CEREBRAL SCLEROSIS.

**Synonyms.**—Sclerencephalia; atrophia cerebri. Tabes cerebri. Atrophy of the brain.

**Definition.**—An induration of the nervous substance, consisting in increase of connective tissue, and atrophy and destruction of the nervous elements, constitutes the condition known generally as sclerosis. The French writers have applied the terms "*Sclerose en plaques disséminée*," "*rubanée*," "*périphériques*," and "*diffus*" to the disease; adopting these names in regard to the character, site, and form of the lesion. Such expressions, while making the nomenclature more exact, imply delicate distinctions which are not always to be made, and do very well only when applied to appearances witnessed after death, but are not so valuable when making a diagnosis before death. I prefer to use the terms "*diffused sclerosis*" of the brain, "*cerebro-spinal sclerosis*," and "*spinal sclerosis*." Even this nomenclature is open to objection, for it is very rare for sclerosis of any kind to be confined to either the brain or cord, though such involvement of the organ not originally affected may be of late date. To confirm this statement I may allude to the ocular symptoms which characterize the early manifestations of posterior spinal sclerosis, or the locomotory defects that are to be seen in some sclerosed conditions supposed to be peculiarly cerebral. I may furthermore add that in all forms of sclerosis there are generally points of induration found after death in both brain and cord. Nevertheless, it is important

<sup>1</sup> Revue d' Anthropologie, tome v. No. 2, 1876.

<sup>2</sup> Journal of Anatomy and Physiology, vols xii, xiv, 1873, 1874.

for us to make distinctions in the manner and origin, course and termination of the various forms of the disease, and we must therefore be contented with an anatomical division.

#### DIFFUSED CEREBRAL SCLEROSIS.

The older writers were in the habit of giving the title "atrophy of the brain" to a condition of that organ which was undoubtedly that which we are now discussing. It is probably one of the most imperfectly understood nervous diseases, and in many instances the diagnosis cannot be made during life.

**Symptoms.**—The cerebral condition, which is tardy induration of an unlimited region, and does not consist in scattered deposits, is a slowly developed morbid state, and is expressed by a train of rather obscure symptoms, the most striking of which are contractions and epileptiform convulsions, impairment of mental power, and various affections of speech. In some cases the conditions date from infancy, and the characteristic feature is want of development of the extremities. In others, a condition of imbecility exists, in which the patient leads almost a vegetative life. One case (No. II.), which I shall relate, was of this kind. Her last years of life were spent in bed, and for a long time there were dementia and unconscious discharges from the bladder and bowels. Some of these cases begin later in life, and the first indications may be either tremor or an epileptiform convulsion, and subsequently various disturbances of motility, such, for instance, as spastic contraction of the muscles of the arm and leg, and the fingers become twisted, deformed, and distorted so as to be useless. Tremor is not rare, and as the disease advances there may be various other symptoms, such as paralysis and muscular atrophy, as well as glosso-labial paralysis. Psychological disturbances are early symptoms, and a species of dementia is rapidly produced.

**CASE I.**—Mary J., the patient, a girl 14 years old, was brought to me during the month of September, 1871. She had been very ill some six years before, and from what I learned from the mother, the attack of illness must have been scarlatina, or some other eruptive fever. Her convalescence was slow, and attended by convulsions of an epileptoid character. She slept much of the time, and seemed dull and stupid. Her memory became impaired, so that her mother was obliged to take her from school, and when allowed to play she quarrelled with the children in the neighborhood, and became so warlike that it was found necessary to keep her at home. When she had suffered for over a year in this way, she began to lose her power of speech, and when she attempted to converse with those who spoke to her she talked in an unintelligible manner; the tongue "seemed to be paralyzed." In 1868 her arms became very weak, and trembling grew violent when she made any manual effort. This loss of power, which was observed more in the right arm, became so great that she was unable to use it in any way whatever. After a year or so the arm became rigid and atrophic, and within twelve months the other arm followed. She is now in a condition of imbecility. She holds her head very far forward when she walks, her chin being

raised. The right pupil is slightly larger than the left. There is ataxic loss of speech, the tongue being entirely out of control, but nevertheless she incessantly tries to talk. Her senses are but slightly impaired, and it may be said she hears well, if we can place any reliance upon the rough tests I made, such as speaking to her behind her back. Her sensibility to pain is not apparently lost, for she gives expression to signs of suffering when she is pinched, but she complains of dysæsthesia.

Her right arm, forearm, and hand are semiflexed and rigid, and the atrophy of the palmar muscles suggests the "*main en griffe*." Her nails are long and thick, and the skin not only of this hand, but that covering the hand and arm of the other side, is blue and cold. The flexors carpi radialis, palmaris longus, pronator radii teres, and other muscles upon the anterior aspect of the forearm were atrophied and contracted, as well as the extensores communis and minimi digiti. This appearance was found on both sides, but more so on the right. When she makes any voluntary movement, the tremor occurs, and it is like that which marks other forms of this disease; that is to say, it is increased by persistence in the attempt. The arms are the only parts affected by the tremor. Her convulsions occur about twice a week.

CASE II.—M. S., aged 18 years, admitted to hospital June 21, 1873. When the patient was fifteen months of age she had her first epileptic convulsions. These, according to her stepmother, have gradually increased in number. At ten years of age she became paralyzed. The paralysis affected her right side, and came on gradually, without loss of consciousness; and it has increased so that at present all the muscles of the extremities, and some of those of the face, are paralyzed. Sensibility is not affected. She has imperfect control of the voluntary muscles, and does not use them readily; and when spoken to does not appear to appreciate what is desired immediately.

Dynamometer: left side 15, right side 19.

The æsthesiometer was not used, as the patient was too much demented to appreciate what was wanted.

Her head is very large, the patient being of ordinary stature. The saliva flows continually from the corner of her mouth, and her complexion is dusky and bad. The muscles are all more or less atrophied. Heart and lungs are normal; no murmurs other than the venous hum of anæmia.

The patient came under my care in June, 1876. She was then in a condition of profound dementia. She had been in bed for some months, and when I examined her I found her conditions to be the following:—

There were no constant ocular defects, no ocular paralysis, and the pupils responded well; but there had been occasional attacks of unconsciousness, attended by nystagmus, when her eyeballs would move from left to right. There was slight paralysis of the buccal muscles, and the mouth was almost constantly open; while a profuse secretion of saliva drooled from the angle of the mouth and over her undergarments and bed-clothes. Her mouth contained partially masticated food, of which there was an accumulation between her teeth and cheeks on either side. Her teeth were very filthy, and the gums tender and bleeding. No appreciative facial paralysis was detected. When spoken to she smiled in an inane manner, but did not attempt to speak. She was occasionally



very apt to cry for several hours at a time, and seemingly without cause. Her position in bed was an exceedingly uncomfortable one; she usually reclined upon her left side, the head drawn down to the same side; and it was agitated by coarse tremors, which ceased when she slept. Her right arm and forearm were drawn to her chest, and likewise agitated by almost constant tremors. Her left arm was also adducted, and the forearm semi-flexed; while the fingers were extended. Tremors of the same character agitated this member. The thighs and legs were drawn up, but did not seem to be quite so rigid as the arms, and there was great atrophy of all four extremities. She passed her excreta unconsciously, and a bed sore had formed upon the left buttock. Voluntary power was absent almost entirely, and I do not remember having seen her change her position in bed from the time I first saw her until her death. Sensibility to pain was very much lost, and reflex excitability was *nil*. Perhaps some of this want of sensibility was due to the horny condition of the plantar skin. She had a great many general convulsions, attended by turgescence of the surface vessels, and nystagmus. She continued in this condition during the year, improving slightly during this time in regard to the number and violence of convulsions, but gradually growing weaker.

*Dec. 26, 1876, 1.30 P. M.* Being fed with stewed meat she had three convulsions in rapid succession, while her mouth was filled with food. Attendant states that she first became cyanotic, but her teeth were so clenched that the nurse was unable to extract the food. As soon as the spasms relaxed, she thrust her fingers in the mouth of the patient, and removed a piece of meat, but the patient was dead.

*Autopsy* 18 hours after death.—No food found in larynx or fauces. Membrane of brain congested and thickened; the gray matter of all the convolutions was of the consistency of the white of a hard-boiled egg.

I afterwards carefully examined the brain, and found patches of advanced sclerosed tissue over the cortex, and throughout the gray and white matter of other parts of the hemispheres. The induration was so general that the brain seemed, as a whole, quite hard and tough. The arteries were diseased throughout, and the calibre of the vessels was quite reduced.

**CASE III.**—This patient presents evidences of cerebral sclerosis, which were evidently of very early origin. The patient is at present in the Epileptic and Paralytic Hospital. Her early history is somewhat meagre. She gives a history of epilepsy, and has attacks several times a week. Her mind is very feeble, and she has attempted suicide several times. The atrophy is one-sided, and there is probably atrophy of the left side of the brain. The following history and table of measurements were furnished by my predecessor, Dr. Janeway:—

E. B., aged 19 years; state single. Admitted to hospital May 1, 1868.

*Examination.*—Head: no facial paralysis or deviation of tongue; no atrophy of tongue; pupils normal, no strabismus; hearing good, as is also common sensibility. Right upper extremity: shoulder-joint is freely movable; elbow cannot be fully extended; hand flexed and extremely pronated; muscles of hand to a certain degree rigid; fingers flexed, thumb not rigid; marked atrophy of entire arm; skin of fingers soft and sodden, but no other changes of nutrition.

*Measurements.*—Middle sternal notch to coracoid process: right side,

4½ inches; left side, 4½ inches. Edge of acromion to external condyle: right side, 10½ inches; left side, 10½ inches. External condyle to styloid process of ulna: right side, 7½ inches; left side, 8½ inches. Apex of acromion to styloid process: right side, 7½ inches; left side, 8 inches.

1st metacarpal bone (index finger): right side, 50 mm.; left side, 55 mm. Metacarpal bone (little finger): right side, 47 mm.; left side, 50 mm. Metacarpal (thumb): right side, 40 mm.; left side, 43 mm.; right index, 65 mm.; left index, 70 mm. Little finger: right side, 53 mm.; left side, 60 mm.

Thenar eminence, thickness of: right, 31 mm.; left, 35 mm. Hypothenar eminence, thickness of: right, 20 mm.; left, 24 mm.

Vertebral prominence to edge of acromion: right side, 6½ inches; left side, 7½ inches. Inner edge scapula to supra-spinal notch, to deltoid: right side, 12½ inches; left side, 14½ inches. Length inner border scapula: right, 5½ inches; left, 5½ inches.

Semi-circumference thorax (4th rib): right, 13½ inches; left, 14½ inches.

Sensibility of right hand normal in every respect. Dynamometer: first trial in left hand, 18; second trial, 10. Hardly any power of right hand, but reflex movements are readily excited in it. Circumference: right arm, 8½ inches; right forearm, 8½ inches; left arm, 9½ inches; left forearm, 9½ inches.

Lower extremities: left, length of fibula, 13½ inches; right, length of fibula, 13½ inches; right calf, 11½ inches; left calf, 12½ inches. Lower edge patella to lower edge external malleolus: right, 13½ inches; left, 13½ inches. Anterior edge inner malleolus to end of great metatarsal: right, 4½ inches; left, 4½ inches. Circumference over heads of metatarsal bones: on right side, 7½ inches; on left side, 7½ inches. Anterior sup. spinous process to lower malleolus: right, 28½ inches; left, 28½ inches. Supra-sternal notch to lower edge of external malleolus: right, 45½ inches; left, 48½ inches.

Sensibility of legs good in all respects. Difference of malleoli as she lies in bed, ½ inch.

**Causes.**—So little is known in regard to the circumstances favoring the development of this disease, that beyond the mention of certain facts of age and sex nothing more can be said in connection with etiology. Women seem to be more affected than males, and we may consider that it is usually a condition that begins in infancy and progresses slowly, or is arrested; or, on the other hand, it may begin in advanced life, and progress more rapidly. In one case which I have seen, syphilis had probably something to do with its development. Scarlet fever or acute diseases of the brain are apt sometimes to leave behind a certain amount of induration.

**Morbid Anatomy.**—Those authors who have made autopsies have found a condition of density of the white matter, the same being shrunken and more firm at the centre of the hemisphere than at the periphery. When a microscopical examination is made, the brain-tissues are found to show appearances which are highly characteristic. The connective tissue will be found to be proliferated, and to present a fibrillated appearance. Corpora amylacea are often present, and we usually find granular deposits

in the blastema. The new tubes are quite changed in character, and are shrunken and attenuated. The axis cylinder may have disappeared, and its place may be filled by a granular substance. The nerve-cells are greatly altered, their prolongations being torn off, and their contents granular. Oil-globules are often found scattered over the field, and sometimes collected about the blood vessels. These vessels are generally much increased in size, and their walls are thickened, and covered by a granular deposit. If the gray matter be the part affected, we shall find an unusual development in the blood vessels.

I have spoken of the involvement of the cranial nerves. It is not uncommon to find at the roots of this nerve a sclerosed point which has involved the nuclei.

**Diagnosis.**—Diffused sclerosis, in its incipency, may be mistaken for cerebral softening, but though the two diseases seem very much alike, the absence of severe pain, and variations of temperature in the latter, as well as subsequent progress of the disease, will enable us to decide; it must be borne in mind, however, that in the great number of cases diffused sclerosis begins in very early life. The congenital cerebral non-development which we sometimes see will be recognized by the absence of tremor, but we must not confuse such cases with those of early intracranial disease where spastic paralysis and increased tendon reflex are conspicuous.

**Prognosis and Treatment.**—The former is excessively bad, and even temporary relief, I think, is out of the question in the great majority of cases. I have never seen a case cured; and if there is any disease of the nervous system that is utterly beyond the reach of drugs, I am confident that it is this. The actual cautery has been used, but, as far as I can learn, without benefit. The treatment of individual symptoms may greatly increase the comfort of the patient, and with this object hyosciamine in doses of from gr.  $\frac{1}{100}$  to gr.  $\frac{1}{25}$  may be given to quiet the tremor or spasm. For the convulsions the free use of ergot does good, while as routine treatment it is advisable to administer the salts of silver or mercury.



## CHAPTER VI.

DISEASES OF THE CEREBRUM AND CEREBELLUM.  
(CONTINUED).

## BRAIN TUMORS.

WHEN the brain chances to be the seat of a morbid growth, whether vascular, or parasitic; homologous, or heterologous, we may be apprised of the existence of such a new formation by a train of symptoms which have no very constant character; or the tumor may even involve a large part of the brain without giving rise to any indications of its presence during the life of the patient. There is no regularity as to the grouping or appearance of symptoms, although the very valuable researches of Hughlings Jackson have enabled us to define the position of the morbid intracranial growths with much greater certainty than heretofore.

**Symptoms.**—We may group the prominent symptoms under the following heads:—

1. Convulsions.
2. Vomiting and vertigo.
3. Headache and cutaneous hyperæsthesia or anæsthesia.
4. Hemiplegia.
5. Paralysis of cranial nerves.
6. Ocular symptoms.
7. Psychical disturbances.

*Convulsions.*—The appearance of convulsions as the only indication of brain tumors has frequently led the observer to make a diagnosis of epilepsy. However, when it is taken into account that there is, at the most, but transitory loss of consciousness—and even this is very rare—during the epileptiform attack, such a mistake is hardly possible. The convulsions may be general or local, and in this place it is proper to refer to the connection between certain cortical lesions produced by brain tumors and consequent convulsions beginning in members which are supposed to have motor centres. Among sixteen cases collected by Hughlings Jackson there were several in which the convulsive seizure began in the thumb of one hand, and finally became general. Cortical lesions were found in the third frontal convolution. In another the epileptiform seizure began in the right cheek, and still another is reported where the right arm was the point of seizure, with subsequent paralysis; and after death a tumor was found in the uppermost frontal convolution on the opposite side. Upon the authority of Bastian<sup>1</sup> and Reynolds, “it may

<sup>1</sup> Op. cit., p. 493.

be stated that convulsions are most common when the disease is situated in the posterior lobes of the brain or in the cerebellum, and least frequent when the anterior lobes are affected." This statement must be considered to apply, however, mostly to those cases presenting general convulsion. Local spasms, which may even be followed by general convulsion, begin in the limb innervated by a psychomotor centre, and are significant diagnostic signs.

Hughlings Jackson considers that psychical disturbances are likewise connected with destruction or injury of the posterior lobes. When the growth is syphilitic, the presence of much headache before the convulsion is the rule. Convulsions may be the first symptoms of tumor, and when they occur in advanced life there is always occasion for suspicion. Several writers have agreed that convulsions and other symptoms are the result of irritation of parts adjacent to the tumor, and that they may vary in appearance and severity in proportion to the local disturbance created by the growth; for this reason convulsions may appear in the most irregular manner. Pain is one of the earliest and most persistent symptoms. It is nearly always localized, and is very intense, especially if the meninges be affected in any way, when it may be combined with muscular twitchings. It is rare for it to subside for an extended period, and then reappear; and in such cases it is highly probable that the growth has either expanded in some other direction, or that the tissues have become accustomed to its presence in the manner suggested by Niemeyer. Pain aggravated at night is highly suggestive of a syphilitic tumor.

Photophobia is sometimes a symptom, and intolerance of noise is a decided feature, while vertigo is produced by very slight irritation, and it has been found in tumors which injure the corpora quadrigemina that this occurs when the patient closes his eyes. Such was noticed to be the case in an example reported by Dr. Duffin. This patient, a man aged twenty-five, presented the following symptoms: A dragging of the muscles at the back of the neck, so that the head was pulled downwards and backwards, unsteady walk, vertigo when eyes were closed, vomiting, frequently slow and irregular circulation, obscured intelligence, double optic neuritis, defective sight, and finally coma. A gliomatous tumor was found which had destroyed the pineal gland, and extended into the optic thalamus. Reeling is commonly associated with vertigo, and is *generally* symptomatic of a growth in the substance of the cerebellum. Symptoms of minor importance are cutaneous anæsthesia or hyperæsthesia, with tingling or formication of the hands or feet. Such anæsthesia may affect the tract supplied by the fifth nerve, while deep cerebral pain may co-exist. This combination is almost pathognomonic, and should be looked upon with suspicion. Total abolition of cutaneous sensibility in connection with cerebral tumors has been studied by <sup>1</sup>Ball and Krishaber. Of

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<sup>1</sup> Tumeurs Cerebrales art. in Dictionnaire Encyclopedique, p. 456.

185 cases of cerebral tumor it was found that sensibility was abolished in but fifteen instances. In seven others it was simply blunted.

Hemiplegia is not an uncommon symptom, and may be sudden when produced by the rupture of a vessel; or of gradual origin, as the result of pressure made upon important parts of the motor tract by a tumor of slow growth. By far the most significant paralyses are those of local origin, connected with local spasms, and these usually indicate a lesion in the cortical motor zone. Paralysis is generally a late symptom, and may begin by loss of power of one member, and afterwards of the other of the same side. By far the most interesting paralyses are those of the cranial nerves, because of their value as diagnostic signs; and not only may the optic nerve be affected, but the auditory, motor oculi, and even the fifth, may suffer an alteration of function.

Jackson and others are of the opinion that those muscles concerned more in the execution of direct voluntary movements are often affected in a greater degree than those which perform automatic movements almost exclusively.

Paralysis of both external recti muscles occurred in one of Jackson's cases, and is, perhaps, one of the most significant indications of the presence of gummata. Lateral deviation of the eyes from the side of the lesion is also a form of cranial nerve paralysis which is by no means a rare symptom. In a case reported by Afanaschiff,<sup>1</sup> where a tumor was found in the right crus, there was dilatation of the pupil and ptosis. Partial paralysis of the face, showing involvement of the seventh, and actual deafness, are not rare consequences of injury sustained by the seventh nerve.<sup>2</sup> When the fifth nerve is affected, as in one of Broadbent's cases, there is generally marked anæsthesia of the region supplied by this nerve, with difficult mastication, deglutition, and articulation. Bed-sores are not met with in connection with paralysis in cases of cerebral tumor, nor do they occur as a result of cerebral pachymeningitis; they are rather the result of hemorrhage into nervous substances. The most important evidences are seen at the fundus oculi, and by some optic neuritis is considered to be a positive sign of brain tumor. Russel,<sup>3</sup> in the description of a very instructive case, details an examination of the fundus. This may be considered a typical example, although the retinal appearances were in an advanced stage. He found "loss of vision complete, neuro-retinitis of both eyes. Right disk comparatively invisible, even its position not clearly distinguishable. Position of left disk indicated by short portion of retinal vessels, which were visible near their point of convergence. Region around the disk in each eye occupied by large irregular patches of hemorrhage, some recent, others undergoing

<sup>1</sup> Wien. Med. Woch., 1870, No. 9.

<sup>2</sup> H. Jackson does not believe that tumors of the cerebrum or cerebellum produce deafness, unless the auditory nerves be pressed upon.

<sup>3</sup> Med. Times and Gazette, July 26, 1873.



absorption. Only very small portions of retinal vessels are here and there visible."

Complete atrophy of the optic disk is generally to be observed in cases where the retinitis has existed for some time.

Hughlings Jackson calls especial attention to the fact that loss of vision is not inseparable from optic neuritis, though complete blindness often does occur. He has seen cases in which there was a double optic neuritis, though the patients were able to read the smallest type.<sup>1</sup>

A very important appearance observed at the fundus, and known as "choked disk" or "congestion papilla," is often produced by brain tumors. In fact, when not a peripheral condition, it is almost always, according to Swanzy,<sup>2</sup> connected with intracranial tumors, hydrocephalus, or meningitis; but when it is produced by these morbid conditions it is usually binocular. "Choked disk" may be caused by a tumor in any part of the brain, whether it be in the cerebellum or cerebrum, and it is not necessary that the optic nerve shall be implicated either at its origin or in its course. Another fact is of importance, viz., that the size of the tumor has nothing to do with the production of the condition, and a small tumor may produce choked disk as well as a large one. The appearance of choked disk is, in substance, the following. The disk may be seen to

Fig. 27.



Choked Disk. (After Leibreich.)

be prominent, the fibres are swollen, and the papillary region is sometimes of a dark reddish-gray, much change of color being due to passive effusion and old hemorrhage. The disk may, in other cases, be of a bright color. There may be some evidences of retinal extravasation,

<sup>1</sup> Royal London Ophthalmic Hospital Reports, vol. iv., 1865.

<sup>2</sup> Signs of Congestive Papilla or Choked Disk in Intracranial Disease. H. R. Swanzy, M. B., F. R. C. S., Dublin Journ. of Med. Science, June. 1874.

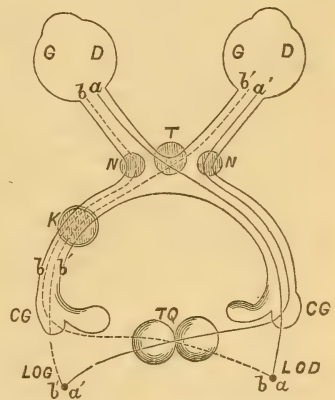
which are not found at any great distance from the edge of the disk, and Albutt<sup>1</sup> says not more than a distance of the radius from the edge. The margin of the disk is concealed by infiltration and by vascularity, which give it a "mossy" appearance. The central radiating appearance resembles very much a scintillating body, while the retinal veins are distended and tortuous, are quite serpentine in their course, and may even be varicose.

I cannot agree with Albutt, who considers the recognition of any prominence of the disk a difficult matter, and I think that this is the opinion of the majority of ophthalmologists.

As interesting features of this as well as other forms of cerebral disease, may be mentioned such ocular trouble as hemiopia and amblyopia. Charcot was the first person to consider the significance of these symptoms and their connection with hemianæsthesia, and he has done much for both neurology and ophthalmology in explaining the direction of the fibres in the optic tracts.

Scheme of the Decussation of the Optic Tracts, according to Charcot: *T*. Semi-decussation in the optic chiasma. *TQ*. Decussation posterior to the corpora geniculata. *CG*. Corpora geniculata. *a b'*. Fibres which do not decussate in the chiasma. *b' a*. Fibres which undergo decussation in the chiasma. *b' a'*. Fibres coming from the right eye, which meet in the left hemisphere, *LO G*. *LO D*. Right hemisphere. *K*. Lesion in the left optic tract, producing right lateral hemiopia. *LO G*. A lesion at this point, right amblyopia. *T*. Lesion producing temporal hemiopia. *NN*. Lesion producing nasal hemiopia.—*Ferrier*.

Fig. 28.



It will be seen by reference to the appended diagram presented by Charcot originally, and modified by Ferrier, that complete decussation of the fibres of the optic nerve (*a a*, *b' b'*) does not take place, but that certain internal fibres (*a' a'*, *b b*) decussate in the optic commissure, while others decussate further back in the tubercula quadrigemina; and that there is a still further complicated arrangement, so that these fibres ultimately centre in the cortex at the pli courbé or angular gyrus. It will also be seen how injury to these fibres or pressure by a tumor or other lesion may produce several varieties of hemiopia. Ferrier describes the production of visual troubles as follows: "Lesion of the left side of the chiasma or of the left optic tract (*K*), will cause hemiopia of both eyes, paralyzing the left side of both retinæ. The external fibres, or those which do not decussate in the chiasma, decussate with their fellows in the

<sup>1</sup> The Ophthalmoscope, etc., 1871, p. 55.

corpora quadrigemina (*TQ*), and so reach the opposite hemisphere; while the fibres which decussate in the chiasma do not again decussate in these ganglia, but pass directly through the corpora geniculata (*CG*) into the hemispheres (*LOG*, *LOD*). In consequence of this arrangement, all the fibres of the right eye reach the left hemisphere, and all those of the left eye the right hemisphere. Hence, lesion of the cerebral centre causes complete blindness of the opposite eye, while lesions lower down, whether in the corpora quadrigemina, corpora geniculata or optic tract, affecting the two sets of fibres before they have run their complete course, cause partial blindness or hemipia of each eye."<sup>1</sup>

Speech is generally involved at some time or other, and psychical troubles of all kinds, but more frequently the æsthenic forms, make their appearance. There is often a condition of hebetude and stupidity which is supposed to symptomatize a tumor in the posterior lobes, or there may be mental decay of a most grave character. Delusions, loss of memory, change of temper, suicidal tendencies, and various perversions of intelligence may occur in any case.

A feature of cerebellar tumor, which I find was also observed by Caton, was the assumption by the patient of the erect position as a means of relief from the nausea and desire to vomit. This author,<sup>2</sup> in reporting a case of cerebellar tumor, alludes to the inability of his patient to regulate his visual co-ordination; and this seems perfectly reasonable when we consider the paralysis of the muscles of the eyeball, and the diplopia, amblyopia, and other disturbances of visual regulation.

The case of Miss F. is in some ways instructive, although it lacks completeness, as it does not contain the report of an autopsy, the patient being still alive (Oct. 16, 1877):—

Miss F., aged 37, U. S. school teacher, was sent to me by Dr. Richard F. Derby, in July, 1876. Seven months ago her present trouble began with weakness of vision, for which she consulted Dr. Derby, of Boston, who adopted Dyerization as a means of treatment. In November, 1876, she began to complain of severe localized headache on the left side of the head. This symptom was constant for three months, and towards the end of this period a gradual hyperæsthesia of the entire left side developed itself, which is now present. It is more decided for three or four days at a time, when there is a lull. There is also strabismus, which attends the paroxysms of acute head pain, which once in a while recur. In December, 1876, there was some vomiting, which did not have any connection with the fulness or emptiness of the stomach. There is no loss of motor power in the upper extremity of either side, but the left leg and foot are rather weak, and there is some awkwardness in progression, the toe dragging slightly. Slight impairment of electro-muscular contractility of muscles of leg and thigh. Dynamometer on left side, 9; on right, 12. Slight ptosis of left eye, occasional diplopia.

<sup>1</sup> Functions of the Brain, p. 168.

<sup>2</sup> London Lancet, Oct. 31, 1785,



Dr. Derby's record of the examination of her eyes: "Neuro-retinitis o. u., with great reduction of vision o. s.; moderate reduction o. d." The patient hears subjective rushing sounds on left side. Is slightly hysterical, and suffers from menstrual irregularities. She gives no history of any traumatism, no blow or fall, nor previous illness. Her mother and father are living, but of decided nervous temperament; paternal aunt and some of mother's connections are insane. Maternal grandmother and her brother died of phthisis. The patient has had night-sweats and some pulmonary symptoms. There is no specific history.

Upon a previous visit she stated that there was great formication in the sole of the right foot. She afterwards went to her home in Vermont, when I lost sight of her, but have subsequently heard of the advance of her symptoms.

The tendon reflex is usually exaggerated upon the paralyzed side; in fact, I have found it to be the forerunner of a hemiplegia, and it may be looked upon as a diagnostic sign, or rather a warning, of what may be expected.

**Morbid Anatomy.**—Without attempting any classification, I will briefly allude to those forms of intra-cranial growth most often met with. Probably that which is most common is *Tubercle*. Amongst young children tubercle is found sometimes in masses of considerable size; and, according to Wilks, the cerebellum is its most familiar seat. It is found as a cheesy accumulation of dirty green color, and very rarely has the grayish appearance of the deposit been found in other parts of the body. These masses are rather dry, and decidedly non-vascular, and if a collection has been arrested in its growth will be found to be encysted, and may be readily removed. If of progressive growth, the limits of the deposit are blended with the surrounding brain-substance, and of a consistency like cold, white glue. Tuberculous masses are rarely single, but generally invade several regions in the same brain, so that it is impossible to give any very satisfactory table which will throw light upon the question of distribution.<sup>1</sup>

Fox, in speaking of Jaccoud's observations, says: "I much prefer Jaccoud's account of these tubercles. They occupy the white and the gray substance equally, and present themselves under the form of small isolated circumscribed masses, varying in number from one to twenty, and

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<sup>1</sup> Grasset\* has classified brain tumors: 1. Those of the *embryonic tissue* (tissu embryonnaire). These are the Sarcomata—*a.* Soft sarcoma; *b.* Sarcoma *nevrologique* (glioma); *c.* Sarcoma *angiolithique* (or psammoma). He considers that the terms glioma and psammoma are improperly used; that the first term suggests more the consistence rather than the character of the tumor. 2. Those of the *connective tissue*, which are—*a.* Myxoma; *b.* Fibroma; *c.* Lipoma; *d.* Carcinoma; *e.* Melanoma. 3. Those of the *cartilaginous tissue*, Chondroma. 4. Those of the *osseous tissue*, Osseoma. 5. Those of the *epithelial tissue*, Papilloma. 6. Those of the *nervous tissue*, Neuroma. 7. Tubercle. 8. Syphilitic Tumors. 9. Parasitic tumors (Hydatids). Aneurism. 10. Abscesses.

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\* *Maladies du Système Nerveux*, Paris and Montpellier, 1878, p. 302.

seldom exceeding the latter. Their volume is in inverse ratio to their number. Pretty often they are the size of a cherry, at other times they scarcely exceed the size of a grain of wheat. As to the colossal masses which attain to the magnitude of a hen's egg, they result from the confluence and fusion of several spots originally distinct."<sup>1</sup>

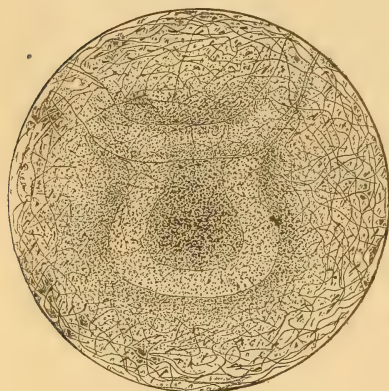
They are sometimes separated from the nervous substance by a sheath of connective tissue and blood vessels. In this connective tissue, which is well filled with vessels, according to Virchow,<sup>2</sup> the new granules are formed, and are impacted with the central mass, and become cheesy. When the process stops, the growth is found to be surrounded by a tough fibrous coat, which is sometimes very hard, and even calcified in old cases.

Ogle<sup>3</sup> has reported a case where the tuberculous mass had broken down, so that it was soft and pultaceous. In the younger subjects tubercle is generally found in other parts of the body. It is quite easy to mistake tuberculous growths for those of a gummatous nature.

*Cancerous growths* in the brain, which seem to affect those of advanced age, take much the same form as they do in other parts of the body. Encephaloid and scirrhus are the commoner forms, though melanomata are occasionally found.

The investing membranes may all be the seat of cancer, but notably the pia mater and the bony walls of the cranium are its starting points.

Fig. 29.



Tubercular Deposit about Vessel.

Fig. 30.



Sarcoma.

In this case the cancerous mass grows inwards, where it meets less resistance, while cancer of the *brain itself* grows outwards. Cancerous masses are occasionally very large, and in one of Russel's cases (to which allusion has already been made) the cancerous mass, which occupied the right parietal region, weighed six ounces and a half. These tumors pre-

<sup>1</sup> Fox, op. cit., p. 151.

<sup>2</sup> Cellular Pathology, p. 523.

<sup>3</sup> Articles in Br. and For. Med.-Chir. Review, 1864 and 1865.

sent the same characteristics they possess in other regions. The encephaloid variety is very vascular; the scirrhous not so much so, and is quite hard. The carcinomatous growth presents the usual appearance of cells contained in the alveoli of a fibrous network or stroma. It may exist alone as an intracranial growth, or coexist with cancer of other organs.

Fig. 31.



Gumma.

Fig. 32.



Psammoma.

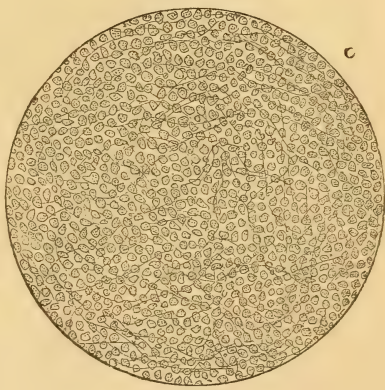
The cancerous growth invades the cerebral substance, though generally the dura mater and the other meninges may be the parts at first affected.

Fig. 33.



Encephaloid.

Fig. 34.



Glioma.

*Syphilis* very often produces changes in the contents of the cranium which are quite formidable. Of diffused infiltration I will not speak, but of those growths known as *gummata*, or "gummy tumors." The meninges and cortex cerebri are commonly the parts which favor the syphilitic



deposits, though deeper regions may very often be invaded by the translucent reddish-gray tumors of specific origin. The interior is sometimes jelly-like and soft, and contains minute red points, while the periphery is hard and fibrous. The tumor proper appears to be separated from the surrounding brain substance by this fibrous covering, though there is always infiltration into the parts adjacent. Syphilitic growths are rarely single, and I have seen a number of them in the same brain. Beneath the microscope the tumor seems composed of round cells about the size of white corpuscles, containing a single nucleus. These round cells occupy the centre of the mass while the outer-portion is composed of a network of connective tissue containing irregular cells. The syphilitic growth may sometimes be mistaken for that of a tuberculous nature. Niemeyer has reminded us, however, "that in syphiloma the passage from the cheesy centre to the broad, grayish-white peripheral zone is very gradual, while in infiltrated growing tuberculi these zones follow each other more closely, and in tubercules that can be turned out they do not exist." The dura mater is very commonly the point of origin. This case, for the history of which I am indebted to Dr. Ryan, was diagnosed by him during life. The patient was in the service of Dr. Mason.

William Browning, æt. 32, native of the United States, boatman, by occupation, married, was admitted to the Paralytic and Epileptic Hospital of Blackwell's Island, on March 13, 1877.

The patient says he has always been a hard drinker. Had been a very healthy man up to seven years ago, when he contracted syphilis, and has since that period been subject, from time to time, to outbreaks of the disease in its tertiary form. Two years ago he had a convulsive attack, which occurred at night; after which he was out of his mind for three weeks. Since that time he has been subject to one or two attacks occurring every month. Since admission, the patient had four epileptiform fits, characterized by clonic spasms, a confused and perturbed condition of the mental faculties, but no distinct loss of consciousness. A premonitory feeling of great terror was always experienced about ten or fifteen minutes prior to the convulsion, and this sense of dread remained for some time after each fit; these seizures being always followed by intense headache and debility, which generally lasted for several days. The patient's sight has failed greatly for the last year; unfortunately no ophthalmoscopic examination was made. His memory, he said, was getting very much impaired, and any mental occupation caused violent headache.

April 28, the date of his last attack, he had been in bed, complaining of severe pains in the head, referred chiefly to the frontal region of the right side. This pain was always greater at night; the patient complained of no other trouble, with the exception of great weakness and anorexia, until about May 5, when slight paralysis of the muscles on the right side of the face was noticed, especially of the orbicularis palpebrarum. There was also a distinct loss of muscular power in the left upper extremity, which was colder to the touch than the right, and the pulse of the affected limb was feeble and compressible. On May 14 the patient became somewhat delirious, and remained so till the time of his

death. On the 17th he began to cough, and expectorated a great quantity of sero-mucous fluid. Mucous and subcrepitant rales were heard over all the anterior surface of both lungs; a change in the pulse and temperature, which had previously remained normal, was now noticed; the former being 130, and the temperature 103°. Herpes appeared on the forehead and lips. On the morning of the 18th, patient was in a semi-comatose condition. Pulse 160, temperature 104°. He died at 2 o'clock P. M. of same day.

*Autopsy* twenty four hours after death. Rigor mortis passing off; body somewhat emaciated; suggillation of posterior portion of body. Old cicatrices (large) over the left tibia, also several smaller ones scattered over exterior and upper portions of body.

**Head:** The dura mater is markedly thickened over portion of the parietal bone of right side adjacent to temporal bone, and is also adherent to a tumor beneath in the brain-substance. On three points on inner surface of parietal bone (right) are spots of necrosis, the size of a dime, which involve the inner table. The dura can easily be separated from the bone, but not from the surface of the tumor. This tumor is three inches from above downwards, and two and one-half inches from before backwards. It is firm, and of a yellowish color. The brain-substance directly beneath it is the seat of softening (inflammation), while beyond this point, and extending in a direct line to optic thalamus of right side, the brain-substance is softened and diffused. The outer border of posterior portion of optic thalamus is in the same condition, while the meninges and vessels are normal.

**Thorax: Lungs.** Bands of adhesion on right side, and a few at apex of left. In the lower lobe of right are numerous spots of lobular pneumonia in gray stage. On anterior margin of right lung some emphysema, and also at apex of left lung. Otherwise both lungs show marked hypostatic congestion and oedema.

Heart soft and flabby. Seat of *post-mortem* decomposition.

**Abdomen:** Liver increased in length; evidences of peri-hepatitis. On surface of liver are seen several old cicatrices, which dip down into liver substance. The parenchyma in patches is softened and fatty (syphilitic liver?).

Spleen increased in size. Capsules thickened in patches; parenchyma diffuent.

Kidneys about normal size. On stripping capsule it brings away portion of kidney tissue. Surface appears granular, and in some points shows lobulation. Section shows tubules swollen, and of yellowish color. There appear to be about normal relations between cortical and pyramidal portions. Pelvis and ureters normal.

Bladder, stomach, and intestines normal.

*Parasitic Growths* (Hydatids and Cysticerci).—Hydatids are always contained in a delicate cyst (except when they occupy the lateral ventricles), and there may be several in the same capsule. The cysts are of variable size, and sometimes attain the magnitude of a fair-sized orange (Reynolds). They are occasionally very large, and the centre of either hemisphere seems to be their common site. Cysticerci, which are very small, and are sometimes contained in cysts, rarely exceed the size of a large marble, but are, however, more often found uninvested, and they

may be from one to several hundred in number. They prefer the cortex, and are often found beneath the pia mater. It seems to me that these would be among the most interesting cases for the observation of irritation of the motor centres; usually, however, there are very slight indications of their presence. In patients who suffer from cysticerci in the brain the diagnosis may sometimes be made by the presence of portions of tænia in the stools, or a cysticercus in the anterior chamber of the lens, which was the case in the example reported by Pollock.<sup>1</sup>

Romberg, while making some experiments, found that the existence of cysticerci in the cerebelli of several sheep accounted for the peculiar rolling convulsions that he had observed.

*Cysts*, which are not the secondary result of softening or hemorrhagic disease, are very rare, and are not usually larger than pin-heads.

*Gliomata*, which are directly formed from the connective tissue, are more common in the posterior lobes and in the cerebellum than in any other locality. The *soft* and *firm* are the two varieties.

\* \* \* \* \*

Amyloid bodies, connective tissue cells and vessels are found to compose these tumors, which may sometimes attain a diameter of several inches. The peri-vascular spaces are filled with adventitious matter, and the calibre of the vessels is very much reduced. These growths may undergo fatty degeneration or absorption. The hard varieties, I think, predominate, and they are very easy to recognise.

*Papillomata*, both of the vessels and meninges, are not uncommon.

*Myxomata*, which Jaccoud describes as having their source of origin from the spheno-occipital suture, are quite rare, as are *Lipomata*. The former are usually of large size, have a gelatinous appearance, and at times are cloudy. The latter consist of large cells filled with fat, and are transparent and shining.

*Sarcomata* may be met with as soft masses, which contain "fusiform bodies, nuclei, and vessels," or else round cells closely packed. They are lobulated, and, when cut, present a pinkish-gray and softened surface, and sometimes contain central fluid. The soft sarcoma, according to Grasset, is found among young children in the deeper parts of the brain, and remains dormant for some time, not giving rise to any symptoms, the cells being usually round ("globo-cellulaire"). With fatty degeneration the tumor may undergo a change, so that it resembles the yellow plates in cerebral softening. It usually has a surrounding vascular network, and is easily separated from the brain-substance.

*Fibrous tumors* are quite rare, but are sometimes met with. Lebert has seen, in one case, seventeen small fibrous tumors upon the ependyma of the lateral ventricle, varying from the size of a pea to that of a small cherry-stone. These tumors are of a white color and globular shape,

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<sup>1</sup> Wiener Med. Presse., 47, 1878.



and they are separated from the healthy brain-tissue by a space in which the vessels are enlarged. They are easily enucleated, and quite hard and dense.<sup>1</sup>

*Aneurisms.*—One of the most interesting and important forms of intracranial growths are those of a vascular character. I have taken occasion to refer to the smaller aneurisms described by Bouchard and Charcot, the so-called miliary aneurisms, which are of minute size; but large aneurisms, arising from such arteries as the middle, anterior and posterior cerebral, basilar, and communicating arteries, may be even an inch in diameter. These, with miliary aneurisms of small size, are generally found to coexist in the brain. Gouguenheim<sup>2</sup> and others have found that aneurism of the basilar artery was much more common than any other form. It is rare, however, that the disease can be diagnosed during life, and but two or three cases have been reported where their presence was recognized by symptoms, and afterwards verified by an autopsy. One of these cases was reported by Coe,<sup>3</sup> another by Jonathan Hutchinson,<sup>4</sup> and a third by Humble,<sup>5</sup> in this case, however, the diagnosis was made by auscultation.

Occasional intracranial growths are the *psammomata* which are found as sandy little bodies scattered over the dura mater, and have a calcareous formation, feel gritty when rubbed beneath the fingers, and may be crumbled. Examined microscopically with a low power they may be found to consist of small, compact, round bodies, imbedded usually in the dura mater.

*Cholesteatoma*, or pearly tumors, which are composed chiefly of cholestérine, stearine, and degenerated epithelium contained in an investing membrane, are occasionally present in the brain. The latter growths are generally found attached to the meninges or cranial bones, and are nearly always superficial.

The literature of intracranial *bony growths* contains much that is interesting. One case reported by Vulpian in the *Archives de Physiologie* was remarkable for the slow development of an exostosis from the temporal bone, which completely penetrated the Gasserian ganglia on the right side. Beyond neuralgia of a severe character, no other symptoms were expressed. I have seen many of these bony growths, some of them even several inches in length, which had existed for years without any mischief being produced. In slow growths there seems to be an accommodation of the brain so that the pressure is rarely injurious, and it is generally not till the exostosis attains some size, and atrophy or softening takes place, that bad symptoms make their appearance.

<sup>1</sup> Anat. Path., vol. ii. p. 71.

<sup>2</sup> Gouguenheim, Des Tumeurs Anévrysmales, etc., Paris, 1866, and also consult Smith, Dub. Jour. of Med. Sci., Nov. 1870.

<sup>3</sup> Quoted by Holmes.

<sup>4</sup> Transactions of the Clinical Society, vol. viii., 1875, p. 127.

<sup>5</sup> Lancet, Oct. 2, 1875, p. 439.

A case which was under the care of Dr. Janeway at the Epileptic Hospital is one of the most remarkable of which I have ever heard, and I append his very valuable record of the *post-mortem* examination.

A. T., aged 42 years; widow; domestic. Admitted to Hospital December 31, 1872. Patient says that fourteen months ago, as she was crossing the ferry, she fell down, and heard people say that some one had had a fit. When she came to, she found that she herself had had a convulsion. During the attack she was perfectly conscious of all that passed about her, and, on arising and attempting to tie her bonnet strings, she found that she could not do so on account of what she says was numbness of the hands or arms.

April 29, 1874. For the past five days she has been very dizzy, and has had headache, and pain in the left side under the breast.

30th. Is in bed. Says "her back feels as if it was breaking in two."

May 1. Is quite weak. Can move her left leg somewhat, but not her left arm; her emotions are easily excited; pulse weak; temperature,  $101\frac{1}{2}^{\circ}$ .

3rd. She lies with eyes half parted, and does not open them fully when spoken to. Pupils normal and respond to light. Answers questions in a slow, whining tone, and with seeming difficulty. Does not draw up her legs when told, but they respond to reflex irritation. The severe pain in her back still continues, and she has some pain under left breast. Pain on pressure in the right iliac region. Bowels free; urine normal; respiration normal; temperature  $100^{\circ}$ . Is somewhat stupid; has great pain in back of her head; eyes half closed; conjunctiva not very sensitive; passes urine and feces in bed.

4th. Sleeping; feces of brown color; urine passed in bed; respiration, 28; pulse, 88. Feces and urine passed in bed during afternoon; tongue dry and coated brown. Only partially protruded tongue when told to. Eyes half closed; seems brighter; respiration, 36; pulse, 100; temperature,  $102^{\circ}$ .

5th. Complains of pain in abdomen; bowels did not move last night; cries when spoken to; pain in back lighter, but in head is sharp. Pulse, 88; temperature,  $100^{\circ}$  at 11 o'clock, A. M. Urine highly colored; no albumen.

10th. Still pain at base of skull. Temperature,  $101\frac{3}{4}^{\circ}$ .

12th. Temperature,  $100\frac{3}{4}^{\circ}$ . 12 M. Temperature,  $99^{\circ}$ ; headache not so severe.

June 2. No headache; cries when spoken to.

6th. Headache not so severe; pain in her back.

10th. Lies with head turned to left. Complains of pain when position of head is changed. Headache is relieved by bromide of ammonium.

19th. Complains of no pain. There appears complete muscular relaxation. Cannot speak without crying.

20th. Patient is rapidly failing. Temperature,  $103\frac{3}{4}^{\circ}$ ; pulse, too rapid to count; respiration very quick; conjunctiva insensible; pupils respond slowly to light.

21st. This morning about the same; can swallow wine. Patient sank gradually during afternoon, and died at 4.30.

*Post-mortem* 18 hours after death.—Heart, liver, lungs, spleen, and kidneys normal. An abscess found in right Fallopian tube containing about 3ij of pus. Rigor mortis not well marked.

*Skull.*—On removing skullcap an outgrowth of bone is noticeable on the right side, near the central line, just posterior to the groove for the middle meningeal artery. The growth is nearly two inches long, and one inch wide; raised about  $\frac{1}{2}$  of an inch from internal surface. The dura mater was pretty firmly attached at this place, and little pieces were left attached to the exostosis. There is another bony projection (small) just back of the middle meningeal artery, at the inferior angle of the parietal bone. Otherwise interior of skull appears normal. The lowest first (1st) is situated just anterior to the fissure of Sylvius,  $\frac{3}{8}$  inch below posteriorly, and  $\frac{1}{2}$  inch from above downwards. Elevation,  $1\frac{1}{8}$ ths of an inch. This has produced a corresponding depression and flattening of the commencement of the lower end of the transverse convolution of the anterior lobe. Two smaller ones are situated one just  $\frac{1}{4}$  of an inch above it, the other  $\frac{1}{2}$  inch above, and about  $\frac{1}{4}$  anteriorly. They are nearly half an inch apart, the posterior being the longer, and about  $\frac{1}{16}$  of an inch in diameter. Elevation,  $\frac{1}{16}$  inch.

Around the first large tumor three small ones exist; the second small one is about one-third of the size of the first. A bridge of new formation connects this with the two already described. At the point of the large exostosis, a number of tumors spring forth from under surface of the dura mater, close to one another, averaging  $1\frac{1}{4}$  inch in diameter. One of these tumors is quite large, and is sunk in a depression in the brain; the depth is  $\frac{5}{8}$  of an inch, and it is an inch long and broad. The brain-tissue around this is in a state of pulpy softening. The diameter of the softened part of brain is two inches, and nearly reaches the longitudinal fissure, extending two inches downwards to within two inches of anterior border of the brain. The *falx* throughout its extent is the site of new formations, some projecting on the right, others on the left; one very large one in front, which is  $1\frac{1}{2}$  inch in length, and has an elevation of  $\frac{1}{8}$ ths of an inch; and another which dips into a depression in the anterior lobe of left side.

The pia mater covering both hemispheres is markedly congested. Tumors are firm, white, and yield only a thin serous fluid on scraping.

**Diagnosis.**—It is a difficult matter, when we consider the great variety and irregularity in the appearance of symptoms, to make always a correct diagnosis. This branch of neurology is undoubtedly the most puzzling, and I am inclined to differ from those persons who consider it possible to determine in the majority of cases the exact location of a cerebral growth. The fact that brain-tumors are very often multiple, and that secondary lesions are produced, is enough to cool the ardor of the most enthusiastic diagnostician. It is possible, however, to sometimes make a very close diagnosis.

We are likely to mistake symptoms of the disease under consideration for those of diseases of an organic character. The common lesions involving a plugging or rupture of the cerebral arteries of the brain may give rise to manifestations much like those produced by intracranial growths.

Paralysis, which is as we know an almost constant symptom of such troubles, differs from that of cerebral tumor, not only for the reason I have stated, viz.: that there are often local epileptoid symptoms in con-



nection therewith, but because the appearance of secondary contractures in the paralyzed limbs is rare. I have found an exaggerated tendinous reflex in the subject of cerebral tumor, but it was never so general as in the other cases, and not attended by spastic rigidity. Then, too, the paralytic phenomena prefer local groups of muscles, notably those of the face, while hemiplegic disorders are peculiar to cerebral hemorrhage, embolism, and thrombosis. Sudden paralysis is rare, though it may occur from a complicating morbid process; but it is not uncommon to find a disappearance and recurrence. Cerebral tumor is rarely preceded by warning symptoms or any adequate cause, except it may be blows upon the head, tuberculosis, or syphilis, but there are many cases with no previous history of any kind. This history of causes is important to bear in mind; for, whether there be inflammation either of an insignificant kind as regards violence, or one of an acute nature resulting in abscess, a history of sunstroke, over-work, alcoholism, or aural disease, may be detected.

Several general diseases may occasionally simulate cerebral tumor,—among them uræmia, narcotic poisoning, heart disease, or even hysteria; but it must not be forgotten that hysterical symptoms are not rare accompaniments of organic cerebral diseases, and often of tumor, so that such cases are not always the subjects of an easy diagnosis.

Localized pain and convulsions, with optic neuritis, cranial palsies, and vomiting, suggest very strongly the probability of tumor.

The localization of cerebral tumors has received very extended consideration during the past few years. In the many cases collected by Jackson we are enabled to make a much closer diagnosis than before his excellent investigations were presented. Ogle's large number of cases are more of interest in the light of morbid anatomy, and as they are several hundred in number, almost every variety of formation is to be found. Quite recently, an excellent article by Petrina, of Prague,<sup>1</sup> has appeared. His directions for localization are so complete that I think it wise to present them, especially as they are based upon a number of cases.

I. *Tumors of the Convexity*.—Clonic spasms limited to single groups of muscles on the side of the body opposite to that of the tumor; no loss of consciousness; incomplete hemiplegia, constant headache, decided vertigo, nervous irritability; amblyopia and disturbances of hearing; circumscribed affection of sensibility. The localization of circumscribed motorial disorders is not definite, and can be only limited at present to the region of the anterior and posterior central convolutions.

II. *Tumors of the Anterior Lobes*.—Frontal headache; the intellectual sphere being involved (? A. McL. H.) there will be often psychical disturbances, with chorea; paresis or hemiplegia (the former more frequently); no disorders of sensibility; general convulsions with loss of consciousness are rare, except when there is deep pressure; visual disturbance and deafness, with anosmia.

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<sup>1</sup> Vierteljahrsschrift fuer di prakt. Heilkunde, cxxxi. 1. 2. Abstract in Journal of Mental and Nervous Diseases.

III. *Tumors of Parietal Lobes*.—Hemiplegia on opposite side preceded frequently by apoplectic attacks; aphasia very frequent when tumor is large enough to compress the third frontal convolution; general convulsions with large tumors; disorder of special sense, except vision, quite rare; impairment of cutaneous sensibility common; frontal headache.

IV. *Tumors of the Occipital Lobes*.—But one of Petrina's cases presented opposite sided paralysis with paralysis of the third nerve on the same side; disorders of intelligence; convulsions, involvement of organs of special sense, cutaneous derangements of sensibility are mentioned by Rosenthal and others as pathognomonic; but are not observed by Petrina.

V. *Tumors of the Motor Ganglia*.—Hemiplegia on opposite side, with loss of consciousness and frequent convulsions; profound cutaneous anæsthesia when the internal capsule is destroyed; sometimes aphasia; *corpus striatum*; complete hemiplegia with loss of consciousness and convulsions; psychic disorders and irritative motor phenomena, such as tremor and choreoid movements; disorders of organs of special sense are rare, with the exception of amblyopia.

VI. *Tumors of Optic Thalamus*.—Extensive motorial symptoms are not constant, and general convulsions or disorders of sensibility are rare. "According as the tumor affects more the bundles of fibres going to the optic tracts of those branching out from the cerebral peduncle, we have sometimes predominating paralytic phenomena in the optic nerve, alterations of the pupil and disturbances of the innervation of the ocular muscles (nystagmus, exophthalmos); sometimes, again, there are the most remarkable vaso-motor anomalies of circulation (striking alterations of temperature, and cyanosis, or circumscribed redness), as the chief morbid symptoms. Pronounced disorders of speech (retarded speech) and of the intelligence are symptomatic only of quite extensive tumors in the thalamus; decided paralytic phenomena are likewise characteristic of simultaneous destruction of the peduncular fibres, or of one of the motor ganglia."

VII. *Tumors in or about the Pituitary Body*.—Somnolence, mental weakness, or apathy; slowness of speech. Amblyopia and amaurosis are common, as well as disorders of other organs of special sense. Rosenthal demonstrated that diabetes is an important complication of tumor in this region.

VIII. *Tumors of the Peduncles of the Cerebrum*.—Vaso-motor disorders and anomalies of temperature; early paralysis of the third nerve on the same side, as tremor, occasional vesical paralysis; opposite hemiplegia with sensory disorders; intelligence unimpaired; optic nerve often involved; involuntary movements of limbs on side opposite to tumor.

IX. *Tumors of the Crus Cerebelli*.—Intense headache and vertigo, involuntary lateral decubitus, rotation of body, one-sided deviation of axis of vision, reeling gait, and tendency to fall; commonly disturbances of organs of special sense. (*Vide* Caton's Case, A. McL. H.)

X. *Tumors of Cerebellum*.—Headache quite intense, and limited to

sub-occipital region, vertigo, reeling gait, disorders of co-ordination; paresis of opposite side of body; convergent strabismus, diminished electro-muscular contractility on sound side of head.

**XI. Tumors of Pons.**—Cross hemiplegia; ocular paralysis (convergent strabismus), lingual paralysis; cutaneous anæsthesia, double or single, dysphagia; disorders of special senses; facial nerve involved; crossed sensory troubles; vaso-motor disturbances; vertigo; increased electro-muscular contractility of parts supplied by the seventh nerve to galvanic current, but not to faradic current.

Greisinger has written quite fully upon the diagnosis of the character of the growth. He considered that convulsion with psychical disturbance, but no paralysis, pointed to the presence of cysticerci, because these parasites infest the uppermost layers of the cortex cerebri.

In one of Jackson's<sup>1</sup> cases (No. 13) the signs of an old iritis enabled him to make a diagnosis of a gumma. Other marks of syphilitic disorder may be taken into account. Nodes, old scars, eruptions of a tertiary character, and alopecia, as well as numerous unmistakable symptoms, such as rheumatism, night-sweats, etc., are confirming points in diagnosis. Aneurism, which is rare in early life, may be suggested by vertigo and subjective noises heard by the patient. In the case reported by Humble a diagnosis was made by the stethoscope. Cancerous tumors are very difficult to diagnose, the age of the patient and the cachectic signs being our only guide, and we are left absolutely in the dark in regard to gliomatous and other non-diathetic tumors, although some of the German writers suggest that a history of injury generally precedes the first named. Tubercle may be suspected after a careful inquiry in regard to the patient's antecedents, and the recognition of the physical signs of deposit in the lungs. Parasitic tumors are generally attended by mental decay, and it has been stated that epileptiform attacks are the first symptoms of such trouble.

**Prognosis.**—Cancerous tumors prove fatal in from two or three months to a year, while syphilitic tumors are occasionally retarded in growth, and the patient may ultimately recover under energetic treatment, though when left alone they rapidly increase in size. I do not believe in the spontaneous cure of aneurismal tumors, and feel disposed to consider any cases of sudden recovery as anomalous. Holmes says in this connection: "We know nothing at present of the diagnosis of intracranial aneurism, so that no treatment can as yet be directed specially to it. And, looking at the very free intercommunication of the four large trunks which nourish the brain, it seems unlikely that surgical measures directed to any one of them would procure the consolidation of an aneurism situated on one of its main branches." The progress of non-diathetic growths is very slow, and the patient may live for many years, and finally die of some other disease. Gliomatous tumors are perhaps less formidable than are others, but after all more depends upon the site of the growth than its size and character. Death is preceded in most instances by coma.

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<sup>1</sup> Medical Times and Gazette, August 1, 1874.



Obernier refers to the increase in growth of cerebral tumors following the excessive indulgence in alcoholic drink, and believes that a debauch may give rise to violent meningitis and death.

**Treatment.**—It has been my practice in every case to place the patient upon an anti-syphilitic course of treatment. The iodide, in increasing doses, until a very large quantity is taken during the day, will sometimes effect a cure. I have given mercury also, but cannot speak so favorably of its virtues. If the pain is excessive, I use the ice-bag, as recommended by Jackson, or the cold water coil of Chamberlain, and find that they give great relief. Hypodermic injections are very useful, and hyoscyamus and belladonna also do good. Galvanism I believe to be useless. Ligature of the carotid has been employed by Coe for aneurismal tumors, and although it was successful in the case he reports, I am inclined to think it is not only a dangerous but an uncertain measure. Humble, in commenting upon this and other cases, speaks of Balfour's plan of treatment, which consists in the administration of large doses of the iodide of potassium. One of the chief indications in the treatment of cerebral tumor is the administration of remedies, and agencies that shall tend to diminish the excessive termination of blood toward the brain, thus cutting off the supply of nourishment as far as possible. A comparatively anæmic state of the brain is better than the reverse. We should caution our patient in regard to the use of stimulants, and should enjoin early hours, abstinence from brain work and rest. Purgatives and local derivatives do much good in certain cases.

## DISEASES OF THE CEREBELLUM.

The cerebellum like the anterior brain, is apt to be the seat of certain familiar morbid processes, and among the more common are *hemorrhage, tumor, softening, atrophy* and the like. Tumor, is perhaps most readily diagnosed on account of the slow development of symptoms, and a certain degree of uniformity in their appearance, but such is by no means the invariable rule.

*General Symptoms of Cerebellar Disease.*—The most conspicuous evidence of trouble is shown in an uneven exercise of motor power, and this has been recognized for many years by all who have had occasion to examine cases of this disease. The defective co-ordination is chiefly shown in grand movements, such as walking, and in certain cases there is a tendency upon the part of the patient to fall backward, while in fact in nearly all there is a reeling, unsteady walk, that by Hughlings Jackson has been compared to the method of progression of drunkards.

This is increased when the eyes are closed, and just as in some forms of other disease, such for instance, as posterior spinal sclerosis, the patient cannot preserve his balance when he has no support. Such troubles result probably from a certain impairment of the harmony of the visual apparatus and the co-ordinating centres, and this in turn undoubtedly arises from derangement of the existing relations between the cerebellar fibres

and the optic lobes. The patients reel and walk with feet spread widely apart. I have repeatedly detected an increase in awkwardness when the person looks up, and when the individual makes any sudden and rapid forward motion, he often staggers and falls backwards. There is rarely paralysis in any form of cerebellar disease until the end, and then it is due to complication of other parts, and is accompanied by rigidity and local spasms. Jackson has repeatedly insisted upon the importance of a symptom of cerebellar disease, and I am able to corroborate what he has said, by personal observation of two cases. He calls attention to violent and forcible flexion as a phenomenon of the convulsions occurring in cerebellar trouble. The head is forcibly drawn backwards, a certain amount of opisthotonus is conspicuous, and at the same time there is extreme flexion of the upper extremities, so that the fists are tightly clenched, the elbows are bent, and there is rigidity, of a very decided character. This condition is observed most perfectly just before death. The tendon-reflex has been found by Sepelli to be present in diseases of this organ.

The oculo-motor symptoms are also a feature of cerebellar affections, and chief among them is nystagmus, the eyeballs being rolled either upwards and downwards, or from side to side. The pupillary changes following an irritative lesion of this organ consists as a rule in contraction which may vary in extent, and it is not uncommon to find a want of response to light stimulation. Amblyopia is apt to occur when the anterior and lower part of the cerebellum is involved, and it may be either double or single, and is a late symptom of decided significance; in a great number of cases optic neuritis is present.

The general convulsions of cerebellar disease are somewhat peculiar, from the fact that there is often rolling of the body which is associated with some fixed deviation of the eyeballs. The experiments of Majendie, Flourens, Brown-Séquard and Ferrier, prove that these rotatory movements with the long axis of the body are constant results of cerebellar irritation, and they occur in the direction of the affected or irritated side, a fact which is of service in localization, as we shall see hereafter.

There is usually a sense of weariness complained of by the patient, though never paralysis, unless other parts are implicated in the diseased process. A prominent sensory disturbance is the sub-occipital headache, which is distressing and painful, and quite common. There may exceptionally be hyperæsthesia of the scalp, though an abnormal modification of the general cutaneous sensibility is rare. <sup>1</sup> Luys, in 100 cases of cerebellar disease, did not find any affection of general sensibility, at least anæsthesia which was uncomplicated, in any of them. He, however, called attention to what has been observed by Rendu <sup>2</sup> and others, viz.: that tactile sensibility is slowly affected in cases of cerebellar hemorrhage. Affections of special sensibility are common enough, and amaurosis may be cited as a symptom of frequent occurrence. It is ex-

<sup>1</sup> Archiv. Gén. de Méd, 1864, p. 580.

<sup>2</sup> Des Anesthésies Spontanées. Paris, 1875, p. 51.

ceptional that we find any prolonged disturbance of the intellect, as we know this region to have little or no connection with the higher mental processes.

#### CEREBELLAR HEMORRHAGE.

The symptoms of this form of disease are difficult to diagnose, because of the liability of the sanguineous effusions to invade other parts in the neighborhood, notably the pons and medulla. Hemorrhage limited to this region rarely produces loss of consciousness, but leaves a train of after-symptoms, which consist of vomiting and ocular disturbances, such as loss of vision, contracted pupils, together with clumsiness of speech, and probably the uncertain gait which has been before spoken of. If there is paralysis, it will be slight and incomplete, unless the outpouring of blood be large, and then important adjacent motor regions are involved.

Carion<sup>1</sup> thus speaks of cerebellar hemorrhage:—"The predominating symptom of cerebellar hemorrhage is general enfeeblement of the muscular system. Hemiplegia is relatively rare; when it exists it is sometimes crossed, sometimes direct. Facial paralysis is exceptional; it involves the orbicular muscle of the eyes, and occurs on the side of the lesion, and it has for its cause the compression of the seventh pair at its point of emergence. The tongue presents a certain degree of asthenia, shown by a weakness in its movements, without deviation. Strabismus, like the facial paralysis, is not observed as a symptom of cerebellar origin; it may occur from compression of some one of the motor nerves of the eye. The conjugated deviation of the eyes has been observed; it always occurs towards the uninjured side as for other parts of the encephalic isthmus. The pupils are sometimes dilated—more frequently contracted; they sometimes react under the influence of light, and are insensible. General sensibility is unaltered even when hemiplegia exists; we barely observe a slight anæsthesia in a few rare cases; hyperæsthesia is still less frequent. Troubles of special sensibility, principally of sight, have been observed, but they are very rare exceptions. The intelligence is generally preserved in all its integrity. Vomiting is scarcely ever absent, and it can rightly be deemed one of the more characteristic symptoms of cerebellar hemorrhage."

Broadbent reports two cases of cerebellar hemorrhage, which are referred to by Wilks. Both cases presented premonitory symptoms of pain, but the other evidences were decidedly negative, and might easily be mistaken for those of other diseases. Both patients died from rupture into the ventricles.

A syphilitic endoarteritis may result in complete stenosis of a cerebellar vessel, so that symptoms of ischæmia are expressed, and become very decided if the closure is complete.

#### ATROPHY (SCLEROSIS) OF THE CEREBELLUM.

Atrophy of the cerebellum is very often met with, and in many cases

<sup>1</sup> Abstract in Chicago Journal of N. Disease, vol. ii. p. 62.



is recognized only at the autopsy. It is as a rule a condition beginning early in life. In those cases I have seen, the atrophy was connected with shrinkage of the cerebral mass. On the same side there was generally some form of mental imperfection or atrophy of one side of the body. Uncomplicated atrophy of one lateral half of the cerebellum I believe to be extremely rare. So far as we are able to judge the symptoms are those which indicate other forms of cerebellar disease, and it is difficult before death to distinguish the condition under consideration from cerebellar tumor of slow growth. There are the disorderly movements, chronic spasms, usually some fixation of the head from rigid contraction of the muscles of the neck, sometimes a series of movements affecting the hands, and which by Sepelli have been described most fully in a case reported by him. In some respects they resembled those of multiple sclerosis, there being a certain amount of irregular jactitation with tremor and a spasmodic expenditure of force. In many cases "atheotoid" movements are presented.

#### TUMORS OF THE CEREBELLUM.

Tumors of the cerebellum may resemble in every respect those found in other parts of the brain, so far as their general structure and topography is concerned. Headache is usually a severe and constant symptom, and is referred to the back of the head, while convulsions are quite severe as a rule, and become more and more violent and frequent as the bulk of the growth increases. Ocular troubles, such as amaurosis, strabismus and pupillary changes symptomatize the presence of growths in this region, and it is common to find decided retinal changes, such as atrophy and hemorrhage. The disorderly movements, which, if once seen, can scarcely be mistaken a second time, are nearly always present, and are connected sometimes with tremor and special paralysis of the cranial nerves. Alteration of the muscular sense and the faculty of localization and sensory perception are quite common. Dr. Webber<sup>1</sup> reports an interesting case of cerebellar tumor with headache, vertigo, vomiting and a species of convulsive attack with aura. There was atrophy of both optic nerves and some unequal anæsthesia of both sides of the body, the left leg and right hand being affected. The patient died suddenly.

"Autopsy.—Brain only was examined. There were a few spots of increased opacity of the pia mater over vertex. Convolutions universally flattened. The ventricles contained a large amount of serum, twelve to fifteen ounces, much of which was lost and not measured. On the under surface of the cerebellum in the median line, between that organ and the medulla oblongata, extending a little farther to the left than to the right, was a tumor; this involved both lobes of the cerebellum and measured about three inches transversely. The medulla oblongata was much compressed and flattened. The tumor contained five cysts: two of which were very large, and two others very small; a large cyst projected anteriorly from above the cerebellum below the corpora quadrigemina. Sev-

<sup>1</sup> Boston Med. and Surg. Journal, April 8, 1880.

eral of the nerves arising from the medulla were thinned, and less white than usual."

In this case, as in many others, the symptoms developed slowly, and the headache before death was much less severe than the beginning, because of the capacity for accommodation to pressure upon the part of the cerebellum, which, as we know, is not readily affected by ordinary mechanical injury. So, too, it would seem that the more serious manifestations of symptoms depend upon invasion of other territories. In most of the cases of cerebellar disease I have been able to investigate, death resulted from softening or injury extending to the floor of the fourth ventricle, or from the bursting of some vessel submitted to dangerous pressure, so that the ventricular cavities become flooded. In this connection may be mentioned a case in which the *post mortem* examination was of great interest.

G. L. C., æt. 26, of nervous temperament; general health good; parents both alive; no nervous tendency; never had syphilis. Four years ago the patient became irritable and morose, and continued so till January, 1873. He then devoted himself to hard study, and rarely took exercise or amusement. Two months afterwards he became debilitated, and had attacks of vomiting, which occurred in the morning, and were relieved somewhat by the upright position. In the following April a loss of steadiness of the lower limbs was noticed. He reeled, and a sudden fright would cause him to fall. He no longer went alone on the street; when he did so, he reeled, staggered, and felt conscious that he was the object of curiosity. His face became congested, and his nose very red, although his habits were good. He went to the seashore, but nevertheless grew worse, and derived no benefit from the change. About this time diplopia troubled him, and he tried various devices to correct this visionary difficulty, such as shutting one eye and looking across his nose with the other, but without relief. In August, violent headache developed itself, and vomiting was frequent. He could not look up or throw his head back without dizziness and pain. Cathartics and local blisters did no permanent good, nor did the bromides.

May, 1875. The patient presents the same symptoms. He is very much troubled by headache, which is paroxysmal. He staggers wildly, and his vision is not improved. On the day before his death he went to see some friends, and on his return complained of a terebrating pain in the back of his head. He went to bed, and slept, under the influence of chloral hydrate. When his wife awoke in the morning, she found him dead. He had evidently died without any convulsions, or she would have been aroused. The night before his death there was some mania, and he shouted words of the different languages he spoke—German, French, Italian—in a confusing jargon.

At no time was there impairment of speech or deglutition; there were never ptosis, deafness, loss of smell or taste. Paralysis was never observed, nor were there convulsions of any kind.

*Autopsy* eight (?) hours after death. The scalp was cut through, and the exposed surfaces were almost black with blood. On removing the bone the meninges were found hyperæmic to a marked degree, the spaces were engorged beneath the arachnoid, and in the ventricles was a large

amount of yellowish fluid, the former being puffed out by the serum under the surface. Nothing unusual was noticed in the hemispheres beyond the hyperæmia before alluded to, and careful slicing of the basal ganglia revealed nothing of importance. The texture of the nervous substance was normal. At the base of the brain a very different state of affairs was found to exist. From before backwards there were evidences of acute inflammatory action, the left side more particularly being the seat of softening. The right crus of the optic commissure was very much disorganized. There was a well-organized membrane, very pink and net-like, which extended over the inferior surface, one band binding down the left root of the optic commissure.

Beneath the lining membrane of the fourth ventricle, at a point beneath the lower and anterior part of the cerebellum, was an effusion, with softening of this organ. This membrane was bellied out, and had evidently produced death by direct pressure upon the *calamus scriptorius*.

At a point corresponding to the middle of the lower vermiform process of the cerebellum was a small hard tumor, about two centimetres in length, one and a half in breadth, and the same in thickness, which, when cut, disclosed a red jelly-like centre, and a hard fibrous exterior, resembling, somewhat, a syphilitic growth. The line of demarcation between the healthy tissue and the circumference of the tumor was very well marked. Beneath the microscope Dr. E. G. Janeway and I found it to be a glioma of the firmer kind, there being a fibrous structure containing the characteristic cells.

After hardening pieces of the cerebellum and the medulla oblongata, I examined them microscopically. The evidences of disorganization of the nervous elements at the nuclei of the vagus were apparent. The nerve-cells were deprived of their processes, and the nerve-tubes were broken. The sections of the cerebellum were made contiguous to the tumor, and here I found considerable thickening of the neuroglia and disappearance of nerve-tissue, while the vessels were very much increased in size.

Strange as it may seem, it would appear as if the progression so far as cure is concerned is not hopelessly bad as the nature of the lesion would lead us to suppose. This is especially true in syphilitic tumor, and I have kept the notes of several cases in which cerebellar tumor was diagnosed and cures were effected in a remarkably short space of time. In one patient the symptoms had existed for ten years, but after the diagnosis of syphilis had been made, and mercurials had been administered, a rapid subsidence followed, and the patient was almost entirely cured within a year.

As a rule, the symptoms of cerebellar tumor of syphilitic origin are complicated by those of meningitis, as tumors of this character start from the investing membrane and grow inwards.

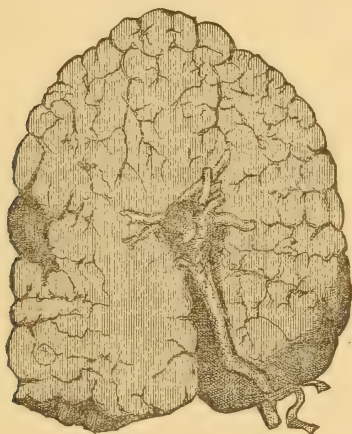
In a few cases of cerebellar tumor I have witnessed mental symptoms, but these are rare. In the case of G. C., an attack of maniacal excitement preceded death. We, not unusually, meet with cases, however, in which there are hysterical complications, just as there are in right-sided organic disease of the cerebrum.



Aneurismal dilatation of the arteries supplying the cerebellum are occasionally met with, and such a case is related by Bristowe.<sup>1</sup>

J. B., a lighterman, was admitted on the 26th of October, 1858, for an attack of acute rheumatism (gout?). No distinct account of the previous duration of his illness was obtained. Five days after admission he complained of severe epigastric pain, and had some vomiting. Shortly afterwards he became comatose, and continued so until his death, which took place on the 2d of November.

Fig. 34a.



Cerebellar Aneurism. (Bristowe).

*Post-mortem Examination.*—There was a considerable amount of serum both on the surface and in the ventricles of the brain; and much atheromatous and earthy deposit in the arteries at the base, and their branches. In the right corpus striatum was a small apoplectic cyst, but in other respects the brain-substance appeared healthy. In the substance of the right hemisphere of the cerebellum was accidentally discovered an aneurism about twice as large as a grain of wheat; it was irregularly fusiform, its parietes were thickened and hardened with atheromatous and earthy deposit, and it gave off several partly ossified branches, each about half a line in diameter. Its anterior extremity was continuous with a thin walled healthy vessel, having between one-third and one-half the calibre of the aneurism itself, and found to be a branch of the right superior cerebellar artery. Gouty indications were found at different points.

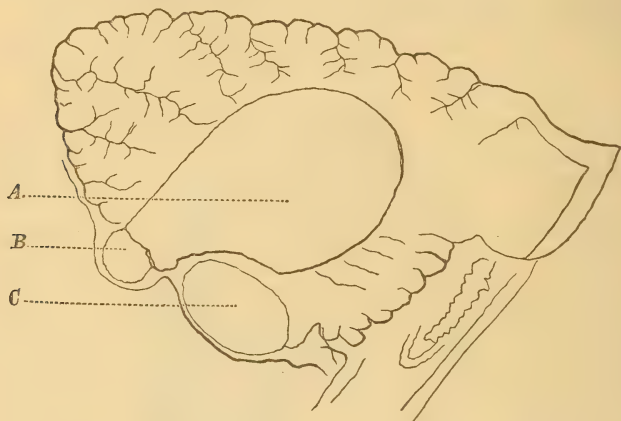
#### SOFTENING AND ABSCESS OF THE CEREBELLUM.

Acute and chronic softening are met with in this organ—and as a result it is not rare to find abscess. Cerebellar abscesses are formed in this way, or depend upon the breaking down of an old clot, as was the

<sup>1</sup> London Pathological Society's Report, vol. x., p. iv.

case in an example reported by Dr. Hughes, of St. Louis,<sup>1</sup> the main symptoms of which are the following:—

“He has a sense of fulness in the head, headaches daily, with intensified pain and throbbing in the occipital region, especially severe in the morning after breakfast. He has a ravenous appetite; vomits often, especially after eating, and has dizzy spells.



A, Abscess. B, Cyst containing serum. C, Organized apoplectic clot.

Before the headaches came on he would sometimes sleep twenty-four hours without waking. When attempting to walk, he often staggers as though he were drunk.

He sometimes hesitates for words to express his ideas, but not enough to be called aphasic.

Three weeks before coming under my treatment, he was much out of his head. He became wild and delirious, and engaged in an imaginary fight with his wife and boy, taking down his gun from over the door to shoot them, saying he must defend himself. He had but a confused remembrance of the fact afterwards. He complains of a sound as of hissing steam in his ears.

\* \* \* \* \*

His sexual appetite was neither absent nor inordinate, so far as we could discover. His mind was clear up to the hour of his death, and a few hours before that event he walked, though somewhat clumsily, about his room. A few minutes before he died he sat up in bed, clasping his hands to his head and crying out with intense pain. He became comatose without convulsive or other premonitions, and fell back on his pillow and in a few moments expired.

On removing the cerebellum, pus and serum escaped through a small opening in the membrane not caused by laceration or scalpel puncture.

The abscess occupies the lower half of the left hemisphere of the cerebellum, extending forwards and upwards, so as to obliterate all traces of the corpus dentatum, and backward and downward, so as to

<sup>1</sup> Journal of Mental and Nervous Disease, October, 1877.

communicate with an apoplectic cell, about the size of a hazel-nut, filled with serum.

This cell extended from the surface through the arbor vitæ arrangement, and opened into the abscess.

The cavity of the abscess was immediately above and contiguous to the organized apoplectic cyst, located just beneath the arachnoid membrane, and occupying the striated structure at the extreme posterior inferior part of the left cerebellar hemisphere, and just within the median line.

This organized blood-clot, though now a little shrunk from long immersion in alcohol, was about the size and shape of a butter-bean.

The apoplectic products did not invade the right hemisphere. The abscess did not implicate any part nearer the middle of the tuber annulare than one and a quarter inches, and of course did implicate the crus cerebelli.

The cavity of the abscess was large enough to envelop a large-sized almond, and was filled with pus.

A careful examination revealed no lesion of the cerebrum.

The weight of the brain, including the pons varolii, medulla oblongata and membranes, was forty-eight ounces and a half. The weight of the cerebellum, medulla and pons, after evacuating the abscess and cell of their pus and serum, was four and one half ounces.

The opposite cerebellar hemisphere appeared neither congested nor in any other manner diseased.

There do not seem to be any very peculiar or *distinctive* symptoms of cerebellar abscess. In many cases, in fact in enough to give the symptom more importance than it receives, there is deafness. The patient is more comfortable in the upright position, and there seems to be more frequent vomiting than in other forms of cerebellar diseases.

The coexistence of aural disease sometimes, either leads us to ignore the cerebellar trouble, or decide at once that the latter is a result of the former, which is not always warrantable. The diagnosis is sometimes made by the ophthalmoscope, and I may refer to Hughlings Jackson,<sup>1</sup> who, in alluding to the importance of this instrument, insists upon the point that very often we have no reason to suppose that there exists any impairment of the visual apparatus, at least so far as the patient's ability to read is concerned.

In a case seen by him the symptoms pointed strongly to aural disease with cerebellar symptoms, but an ophthalmoscopic examination revealed double optic neuritis, though there was no cranial nerve paralysis. She saw perfectly, though her retinae were the seat of disease. A *post-mortem* examination revealed an abscess in one-half of the cerebellum of great size.

**Pathology and Morbid Anatomy.**—The results of much experimentation show that injury or disease of the cerebellum is followed not only by special symptoms, but by others indicating disturbance of the conjoined function of the cerebrum and cord, and that as this organ

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<sup>1</sup> Remarks upon the routine use of the ophthalmoscope in Cerebral Disease, p. 16.



is the seat of the so-called "muscular sense," there is an impairment or abolition of this function as well. The cerebellum seems to play a regulating part, if such an expression can be used, for there are a number of indirect disturbances in the functions of the anterior brain which are produced. Bastian refers to the liability of the cerebrum to suffer in such cases by reason of mechanical vascular interference. The *venæ galeni* which empty into the straight sinus are subject to pressure when the middle lobes are affected. This in some measure accounts for the indirect production of hemiplegia in a number of cases of cerebellar disease while in other forms in which there is very decided destruction of the cerebellum, no real paralysis occurs but simply a "weakness." When hemiplegia occurs it is sometimes due to pressure upon the medulla, and is irregular in its production. When one lateral half of the cerebellum is the seat of injury, we have hemiplegia upon the same side of the lesion, "an effect really induced by the pressure which such lesion occasions upon the corresponding side of the medulla oblongata."

Irritation of the cerebellum by means of electricity has been found by Hitzig<sup>1</sup> to result in a peculiar train of phenomena. A galvanic current passed through the head, the electrodes being placed upon either mastoid process, produces immediate dizziness and a disturbance of equilibrium, depending upon the position of the *anode* and *cathode*.<sup>2</sup> The passage of the current from the right to left, the anode being placed upon the right mastoid process, causes a vertigo in which external objects move from right to left, and according to Ferrier, when the subject closes his eyes he feels as if he were being twirled from right to left; a contrary state of affairs occurs when the poles are reversed. The eyeballs are directed to the side of the body towards which objects seem to move.

So far as the loss of equilibrium is concerned, it has been found that the most active expressions of disturbed motility follow immediately after the injury or occurrence of the lesion, and Ferrier says take place as a result of the "sudden derangement of the self-adjusting mechanism on which the maintenance of the equilibrium mainly depends."

It would appear from the records of ninety-three cases brought together by Andral, and a dozen or more cases collected by Hughes, that a very considerable destruction of the cerebellum may take place without any conspicuous alteration of functions so far as motility is concerned; and it would also appear that the morbid processes characterized by hypertrophy or tumor are those in which the most decided phenomena are presented, and presumably as a result of pressure made upon other parts. From the physiological experiments of Ferrier and the clinical observations of Bastian and others, we may roughly approximate as follows the localization of cerebral disease:

*Injury or Disease of the Middle Lobes.*—Pitching forward of the body.

<sup>1</sup> Quoted by Ferrier, page 106.

<sup>2</sup> Anode: positive. Cathode: negative.

Affection of vision due to irritation of optic lobes. If the upper part is affected: nystagmus to the right horizontally—if the lower, in the reverse direction. Symptoms indicative of cerebral pressure due to ventricular dropsy. Increase of sexual power (?).

*Injury or Disease of Lateral Lobes.*—Rotatory movements towards diseased side (Schiff, Ferrier, Hitzig, and others). In cases of limited disease there is a tendency to fall towards side of lesion, rolling of eyes upwards, inwards, and towards side of lesion. Hemiplegia, perhaps, and when found, it is more marked in the leg than arm, and is accompanied by loss of sensation. If one lobe is affected, there is rarely decided loss of power, but unsteadiness and weakness. No affection of speech or deglutition.

*Injury or Disease anterior of Anterior Region.*—Vertical nystagmus, complicating cerebral disturbance.

The *morbid anatomy* of the cerebellum presents a large field for study; and Pierret, Meynert, Fischer, and Sepelli have recently written a great deal that is valuable. Disease of this organ presents ultimate textural changes that differ but slightly from those which affect other parts of the brain. The commissural fibres are often found to be the seat of degenerative changes which may extend to the cerebrum and the cord, and it is not uncommon to see atrophy and sclerosis of other organs in the vicinity in connection with morbid processes in the cerebellum itself. In some cases the pons is greatly diminished in size, while it is to be observed in others that the cord is the seat of secondary degeneration, as a result of downward extension of cerebellar disease; but this is not nearly so common as when it follows cerebral disease. When such secondary degeneration exists, it may be explained by reference to the anatomical relation of the series of fibres that pass either across the median cerebellar peduncle or through the medulla to enter into the formation of the anterior and lateral columns. It follows in certain cases, therefore, that secondary contractions are to be met with; and in a patient who died at the Hospital for Nervous Diseases, there was besides atrophy of the cerebellum and cerebrum, a hemiplegia with secondary contractures and sclerosis of the cerebellar peduncles. In many cases the cells of Purkinje will be found to be altered, having undergone granular changes. Softening is common, and this may be readily inferred when we take into account the rich vascular supply of this organ.

In different cases of abscess of the cerebellum, the size of the purulent collection will vary greatly, and frequently one-half of the organ is found to be the seat of a cyst filled with pus. As in Hughes' case, these cysts often follow old hemorrhages. <sup>1</sup>Fox presents a case in which cerebellar abscess existed together with small abscesses of the cerebrum and lungs, and another in which a large abscess in the central part of the left hemisphere of the cerebellum existed with distended ventricles. In neither of these cases was there any apparent cause.

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<sup>1</sup> Pathological Anatomy of Nervous Centres.

The vessels liable to rupture are the inferior cerebellar arteries, the posterior being a branch of the vertebral, and the anterior a branch of the basilar. The vessel however most frequently found ruptured is the superior cerebellar artery, usually at a point where it gives off a branch to supply the rhomboidal nucleus. The arrangement of the vessels is double and there is free anastomosis. As in other parts of the brain, the hemorrhages into the gray substance are the most extensive, and this is especially the case when the superior cerebellar artery is ruptured.

Atrophy of the cerebellum is usually a congenital state, although it may follow low inflammatory processes, or be due to osseous deformities, as in the case cited by Otto. Meningeal thickening may induce atrophy by pressure.

As to cerebellar tumors, we find that glioma prefer this seat, though tubercule is by no means rare.

**Diagnosis.**—Cerebellar disease may be confounded with several other forms of trouble producing disorders of motility. Chief among these are anomalous varieties of *locomotor ataxia*, in which head symptoms are marked. There is never, as I have stated, any disappearance of the tendon reflex, though atrophy of the optic nerves may be present in both diseases. I have repeatedly met with cases in which the diagnosis, so far as the gait was concerned, was extremely difficult. In cerebellar diseases there are none of the sensory disturbances so marked. The neuralgic pains in the lower extremities and anaesthesia are therefore absent, as are the *crises gastriques*. The differential diagnosis between *cerebellar hemorrhage* and that in the cerebrum is not so difficult, for there is rarely any loss of consciousness, unless the hemorrhage is sufficient to flood other parts. The symptoms are characterized by their regularities in their grouping. A point previously stated should be borne in mind, and this is, that the paralysis—if it be present—is much more profound in the lower extremities, and that facial paralysis is rare, a point insisted upon by Bastian. In the various forms of cerebral sclerosis the diagnosis is quite difficult; in fact, the cerebellum is rarely the seat of limited sclerosis, as in cases reported by Charcot and Bourneville other parts of the brain were affected as well.

In a great many cases disease of the cerebellum gives rise to convulsions, which are mistaken—and not without reason—for epilepsy. They are irregular, however, and connected with such marked tonicities that they need not mislead. Moreover, they often occur without loss of consciousness, and are connected with vomiting and nystagmus, and are always *bizarre* and rotatory.

**Prognosis.**—The most faithful and intelligently selected treatment avails but little in cerebellar disease, except in certain exceptions. Syphilitic disease, in this region I have found, as I have before said, to be much more effectively combated than when it involves other parts of the brain. The progress of cerebellar disease is so slow, and, as a rule, is so rarely attended by serious symptoms as to be less alarming to the patient and physician than where the pathological process involves some other region.



**Treatment.**—The management of these cases is very much like that which should be followed in general cerebral disease. The iodide of potassium in large doses, arsenic in the form of Fowler's solution, or the bi-chloride of mercury may be given a thorough trial. Counter-irritation by means of a seton, or frequent cauterization of the neck, should be resorted to as well.

In a case of syphilitic origin I had the pleasure of witnessing a very rapid disappearance of symptoms when the patient was submitted to systematic inunction with mercurial ointment. For the relief of the intense headache, the ether spray to the occiput, or ether applied on cloths to the head, as recommended by Dr. Hughes, affords great relief.

## CHAPTER VII.

## DISEASES OF THE SPINAL MENINGES.

## SPINAL MENINGITIS.

## ACUTE PACHYMEINGITIS.

THE investing membranes of the spine may be the seat of chronic or acute inflammation, together or singly, though there is generally a certain amount of coexisting myelitis, and consequently the meningitis is not an uncomplicated condition. In exceptional cases, however, the dura mater may be affected, and the resulting affection is known as *Spinal Pachymeningitis*; or the pia mater and arachnoid in other cases are the seat of such inflammation; or the three membranes may be together involved.

## INFLAMMATION OF THE SPINAL DURA MATER, OR SPINAL PACHYMEINGITIS.

Michaud<sup>1</sup> has given the name *external pachymeningitis* to the form which results from pressure made by diseased vertebræ, and coexisting with Pott's disease, while other varieties have been described as *internal hemorrhagic pachymeningitis* (Meyer<sup>2</sup> and Schuberg<sup>3</sup>) and cervical hypertrophic pachymeningitis (Charcot<sup>4</sup>). The form described by Meyer is almost identical with that which involves the cerebral dura mater, and in which there is thickening and encysted clots. As the name indicates, the form described by Charcot is confined chiefly to the cervical portion of the spinal dura mater.

## ACUTE AND CHRONIC SPINAL MENINGITIS.

**Symptoms.**—This disorder, which commonly involves all three membranes, is generally ushered in by a chill, followed by elevation of temperature; a hard, full pulse, and excruciating pain. This pain is increased by any movement the patient may make. He tries to relieve his suffering by changing his position and by keeping quiet, so that muscular rigid-

<sup>1</sup> Sur la Méningite, etc. Thèse de Paris, 1871.

<sup>2</sup> De Pachymeningitide, etc. Dissertatio inaug. psych. Aug. Meyer. Bonnæ, 1861.

<sup>3</sup> Vich. Archiv., t. xvi. p. 481.

<sup>4</sup> Leçons sur les Fonctions du Sys. Nerveux, fas. 1, part 2, p. 243, etc.

ity, which is semi-voluntary, is often mistaken for a tetanic spasm. Pain darting along the spinal nerves adds all the more to his misery, and his legs are forcibly drawn up. Hyperæsthesia of the surface is generally present, and reflex excitability is nearly always exaggerated in the earlier stages. The head is sometimes drawn backwards by contraction of the post-cervical muscles, and the appearance is presented which is so well marked in cerebro-spinal meningitis. Should the meningitis be general, or extend upwards, the intercostal and phrenic nerves are finally involved, and asphyxia and death result. The tendency in many cases is towards chronicity, and very often there are secondary affections of the cord from pressure. The bladder and rectum frequently suffer to such a degree that involuntary discharges of urine and feces result, but the former sometimes escapes the involvement. Should the disease become chronic, it exists in a modified form, the pain being less severe, and the contractions of the limbs more marked. The skin is cold and hyperæsthetic, and reflex excitability is present to an extraordinary degree, the slightest prick of a pin being sufficient to cause violent retraction of the limbs. The muscular power is greatly reduced, so that the individual may be unable to take any exercise. The bladder trouble is much more marked than in the acute variety, and the patient may find it necessary to empty his bladder every few minutes. Obstinate constipation, distension of the bowels by wind, and gastric disturbances, are accompaniments. If the cord is involved, there may be presented symptoms of meningo-myelitis, and then paralysis of motion and sensation becomes marked, and the muscles undergo atrophic changes.

The case of Mr. J. E. is instructive. He is a great sportsman, and up to four or five years ago was often exposed during his hunting excursions. Four years ago, during one of these, he lay for several hours in a "battery," shooting ducks. The weather was cold, and he was directly exposed to a drizzling rain. On the same night he was seized with a chill, which lasted for nearly an hour, and, supposing he had "caught cold," he drank altogether nearly a tumblerful of whiskey. During the night he became feverish, complained of pain in the back, vomited, and was delirious throughout the next day and the two following. His pain was excruciating, and the slightest jar of the bed caused him intense agony. At the end of fourteen days he was moved upon a mattress to the nearest boat, and from thence to the railroad, and was carried to his home by easy stages. For a month or so after, he was confined to his bed, the pain gradually becoming less intense, and his strength returned by degrees. He presented himself to me with the history I have just detailed. For the past year he has had spinal pain, which he refers to the last dorsal and upper lumbar vertebrae. It is constant and worse at night, and increased by pressure. There is gastrodynia, and pains down the back of the thighs, which seem to increase after exercise. He complains of loss of power in the legs, and cannot walk more than a block or two without being greatly fatigued, and at night his legs are jerked up during sleep. For the past year he has had great distress and discomfort, as he cannot hold his water, and is obliged to empty the bladder every few minutes. His bowels are so constipated that he finds it

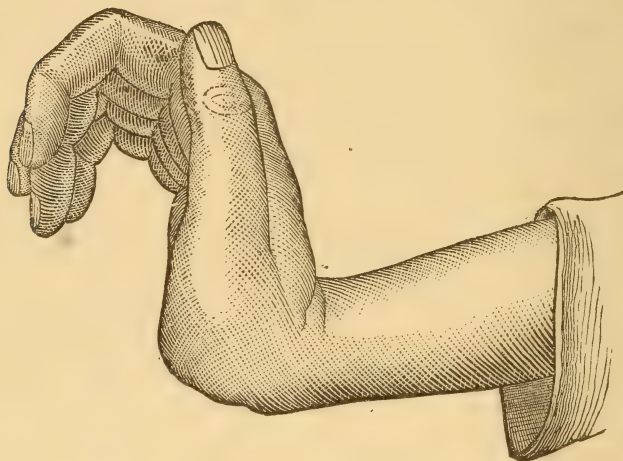


necessary to use an injection every night. Examination revealed pain upon pressure over the two lower dorsal vertebræ, analgesia and anæsthesia of the cutaneous surface of the posterior region of thigh. The glutei muscles, as well as the adductors of the thigh, were much reduced in size, and did not contract as powerfully as did those in the neighborhood when subjected to electrical stimulus. His abdomen was tympanitic and greatly distended. He had become despondent during the past year, and neglected his business. In addition to the pain, loss of power, and the other symptoms I have enumerated, there has been a sense of abdominal constriction at the level of the painful point. Damp weather aggravates the pain, and he has periods of improvement, when he goes to Florida or some other warm region.

#### SPINAL PACHYMENINGITIS.

**Symptoms.**—The forms of pachymeningitis cannot be during life separated as a rule. There may be no acute stage whatever, but a gradual appearance of symptoms indicative of slowly developed pressure upon the cord. The form described by Charcot<sup>1</sup> runs its course in five

Fig. 35.



Deformity of Hand in Cervical Pachymeningitis (Charcot).

or six years, and the cervical enlargement of the cord is the part which suffers the most. Pressure is made upon the cord itself, and upon the nerve-trunks, so that partial or total loss of function ensues. There is a painful stage, the *première période* of Charcot, which lasts several months, the pain being intense at the back of the neck and in the upper extremities. With these pains there is rigidity of the upper extremities, and the head is drawn backwards and downwards in the manner I have before described. There are in addition formication and disagreeable sensations in the upper extremities, twitching, and some paresis, which ultimately

<sup>1</sup> Op. cit.

increases, so that the individual retains but little power. Charcot has observed eruptions of bullæ and pemphigus as evidences of lowered vitality. After this period there is atrophy of the paralyzed muscles, particularly those innervated by the ulnar and median nerves, while those which are supplied by the radial escape the atrophic change, and deformity often results which somewhat resembles the *main en griffe* of progressive muscular atrophy, diminution and loss of electrical excitability. The preceding cut from Charcot represents the appearance of the hand in this condition.

Contractions of the paralyzed muscles ultimately follow the paresis, and the skin becomes decidedly anæsthetic, so much so that a pin may be inserted without any expression of suffering from the patient. It is very rare for the lower extremities to be implicated, and the medulla seems to escape the effects of the disease, consequently troubles of deglutition or respiration are rare. The hemorrhagic or internal form of pachymeningitis runs a most irregular course, but the complicating spinal affections are apt to be much more marked than in the last-mentioned variety. The indications of internal pachymeningitis are throbbing pain in the back, sudden paralysis, and the other symptoms to which I have alluded. The disease is connected with hemorrhages, and consequently there are at intervals accessions of fresh symptoms.

In a large number of cases the symptoms may be due, in the first place, to pressure from diseased or fractured vertebræ, and pronounced pain of a somewhat local character is a prominent initial expression of trouble, and this will be followed by other symptoms, at first comparatively localized, but eventually, the pain will extend, and descending or ascending expressions of compression of the cord will be manifested.

The large number of cases which were known as "syphilitic paraplegia" some years ago include many examples of chronic syphilitic pachymeningitis, which were then recognized as the result only of myelitis. The progress of the disease is much more slow than in other forms, and the patient lasts a very long time, and is sometimes quite cured by appropriate anti-syphilitic remedies. The acute zymotic fevers are not rarely followed by pachymeningitis, the following case being an interesting example of this occasional sequel of typhoid fever:—

Two years ago Capt. S. recovered from an attack of typhoid, and with convalescence he gradually lost power in the right hand, right leg, left leg, and left hand, in the order I have named them (this is his statement). Preceding these conditions there were shooting pains running down the spine and around the body. He was paraplegic two months afterwards. During this time reflex movements were easily provoked. "When my feet came in contact with the foot of the bed, if the cold wood touched them they would fly up." He evidently had the contractions which are so clearly symptomatic of meningitis, and there was some constipation, but no bladder trouble except atony. His neck "felt stiff," and he was occasionally dizzy. The loss of power in legs has gradually returned.

*Present condition.*—The patient walks fairly, with no apparent impediments. The skin is slightly hyperæsthetic; no atrophy of any muscles; has good muscular strength; there is slight tenderness produced by pressure over the vertebræ between the scapulæ; muscular tension at back of neck, and some pain with movement; slight distension of abdomen by flatus (he says this is a constant symptom); bladder and bowels in excellent condition; some very trivial effort required to urinate; no headache, but dizziness caused by looking upwards; no loss of power in hands or arms; no constricting band; patient can stand with eyes closed. Co-ordination of delicate muscular acts unimpaired; there are no twitchings at night left. I suggested the propriety of giving iodide of potassium in addition to ergot, which he had taken before. I also recommended the actual cautery.

One of the characteristic symptoms of all forms of spinal meningitis is the rigidity of the spine, and there is an increased excitement of the tendinous reflexes which may be unilateral or bilateral. In the contracted limbs the percussion hammer produces a very energetic series of motor phenomena. The contraction of the muscles are usually aggravated when some voluntary effort is made to overcome them, but the fingers of the patient may be often passively extended when his attention is diverted.

*Causes.*—According to Grisolle,<sup>1</sup> spinal meningitis is much more common among men than women, and three-quarters of the patients are men; and Calmiel considers it to be of much more frequent origin before the thirtieth year than afterwards. Cold and intemperance favor its appearance, but in the great majority of cases, it is of spontaneous origin, and has occurred in epidemics, at least so say the earlier French writers.<sup>2</sup> In 1837 an epidemic appeared at London, Versailles, Avignon, Metz, and Strasburg, and there were no atmospheric causes nor any influences discovered which could account for its appearance. It is probable, however, that the form of meningitis was cerebro-spinal, with the history of which we are now familiar. Alcoholic over-indulgence, syphilis, and injury, or vertebral disease, will account for the affection in some cases. Like locomotor ataxia it very often occurs among seafaring men who have fallen overboard, or have been obliged to stay aloft in damp, cold weather. Pott's disease has generally been supposed to have little to do with the etiology of the disease, but my own experience and that of professional friends who have had much to do with this class of cases, convince me to the contrary. In a case of this kind where I was enabled to make an autopsy, I found great thickening of the spinal dura, with fibrinous deposits beneath that membrane and the bone, as well as some involvement of the nervous substance proper, which consisted in atrophy. Fractures of the spine, sometimes unrecognized, are attended by so much injury of these membranes as to give rise to symptoms which may be either sup-

<sup>1</sup> Op. cit. vol. i. p. 436.

<sup>2</sup> See articles in *Mémoires de l'Académie Nationale de Méd.*, t. x., *Revue Médicale*, and *Gaz. Médicale*, 1842.



posed to be due to myelitis or simple concussion, but which are undoubtedly occasioned by an unrecognized fracture. Such a case has been reported by Mr. Hutchinson, in which the individual jumped from a height, alighting on his feet.

**Morbid Anatomy and Pathology.**—The simple forms of spinal meningitis, that is to say the acute forms, present all the appearance of violent inflammatory action which we witness in cerebral meningitis: injection of the pia mater, serous or purulent effusions, together with infiltration of adjacent cellular tissues, more posteriorly than anteriorly, and perhaps some evidence of myelitis, but ordinarily the cord is healthy if the disease be uncomplicated. The region affected is more apt to be at the upper part of the cord, but there may be inflammation of the meninges covering the dorsal or lumbar portions as well. It may be circumscribed, as the result of pressure from displaced vertebræ, or fracture, and this limitation is more characteristic of pachymeningitis. The different membranes may be adherent to each other, and connected with the cellular tissue in the vertebral canal. New growths beneath the dura mater are not common, but may be found sometimes between this membrane and the bones. In cervical pachymeningitis there is great thickening, and in old cases the nervous matter is compressed to such a degree that it is atrophied, and may be found to be hardly two-thirds its normal size. A lamellar arrangement of the dura mater exists, which is like that seen within the cranium, and the other membranes may be quite undistinguishable from the dura mater, and consequently the cord will be found encircled by an almost homogeneous, tough, and thickened envelope. The cord, when the thickened membranes are removed, often presents an irregular contour, evidence of sclerosis being common. The lateral and posterior columns seem to suffer most. In the hemorrhagic form, there may be discovered encysted blood-clots which resemble those found in cranial hemorrhagic pachymeningitis. The nerve-trunks within the vertebral canal will be found to be covered by the same dense tissue, and the peripheral portions of the nerves are often atrophied. Syphilitic inflammatory changes, alluded to by Buzzard,<sup>1</sup> are sometimes present, with gummatous growths in the nerves proceeding from the cord.

The following case illustrates the morbid anatomy of meningo-myelitis of a quite extensive character :—

*Idiot; Chronic Spinal Meningitis; Lobular Pneumonia; Circumscribed Acute Interstitial Nephritis; Chronic Cystitis.*—D. A., æt. 26, admitted June 22, 1877. No previous history of the patient could be obtained, except that she had been an inmate of the almshouse for three years previous to admission, where she was confined to bed entirely. On admission patient was very much emaciated; legs and thighs flexed. She was unable to talk, but almost continually screeched, especially at night. Two days before her death she had a slight diarrhœa. On morning of June 28 had elevated temperature, rapid pulse, and cough. Chest could not

<sup>1</sup> Syphilitic Nervous Affections, p. 70.

be satisfactorily examined, as she would not keep quiet. Moist rales were heard over entire chest. Patient became worse during the day, and died at 4 o'clock A. M., June 29, 1878.

*Autopsy twelve hours after death, made by Dr. Maxwell, the Curator.*—Rigor mortis present; body small, and very much emaciated; thighs flexed and adducted, and the legs upon the thighs, and contracted. Feet œdematous. Bed-sore over sacrum and nates. Fingers and thumbs are flexed; the cranium small; round, low forehead; hair dark; complexion brunette; eyes brown.

*Head.*—Bones: calvarium circular; antero-posterior diameter six inches; deep Pacchionian depression on right side. Dura mater and sinuses normal. A little over three ounces of fluid in subarachnoid space. Pia mater over the convexity meshes is markedly elevated by œdema, and is opaque in latter situation; it is also abnormally adherent over convexity, and in Sylvian fissure. Weight of brain and cerebellum 22 ozs. Externally shows nothing except that the sulci are wide. Lateral ventricles are moderately dilated. Ependymæ appear normal. Cerebellum weighed  $1\frac{1}{2}$  oz. Brain-substance of cerebrum and cerebellum, gross appearances normal.

*Spinal Cord.*—Adhesion in cervical region, between dura mater and wall of spinal canal, so firm as to require section for its removal; also another point in dorsal region. Adhesions between opposed surfaces of arachnoid in cervical region quite firm and general on the posterior surface; on anterior surface scattered filaments. On posterior surface of dorsal region a few filamentous adhesions. Dura mater in cervical region is appreciably thickened, especially the upper two inches. Pia mater corresponding with these adhesions has brownish appearance, and is thickened. Veins of cord are filled. Nearly all dorsal portion of the cord is soft to the feel. Throughout cervical region the posterior and right lateral columns are to the feel firm and normal; have bluish-gray color, with yellowish streaks. The dorsal portion of the whole cord markedly softened. Lumbar region and caudæ equina, to gross appearances, show nothing marked. Dura mater surrounding vertebral foramina is thickened and adherent to sheaths of upper four or five inches of cervical nerves. Posterior long fissure of cord of the dorsal region obliterated by firm adhesions of pia mater.

**Prognosis.**—The patient's chances are sometimes good, even in the chronic form. Charcot<sup>1</sup> has cured one case of cervical pachymeningitis, and doubtless others have been equally successful. In the great number of cases, however, a fatal termination is the rule. In the acute form death may occur in six days, but Tourdes and Chauffard have observed cases in which this termination did not take place till the fortieth or fiftieth day. In acute purulent meningitis the pus may make its way out, pointing externally, or forming an abscess in the muscular tissue of the back. Champion has seen a case of this kind in which the purulent contents of the vertebral canal found passage through at the third lumbar vertebra, and formed an abscess in the spinal muscles. This, however, is exceptional. When the disease results from Pott's disease, or some other vertebral affection, it is perhaps possible, by mechanical treatment, to improve or

<sup>1</sup> Op. cit.

cure the patient; and syphilitic forms, of course, are generally amenable to treatment. Death may occur from exhaustion, and is preceded by the formation of bed-sores, and evidences of a typhoid state.

**Diagnosis.**—It is necessary to diagnose spinal meningitis of the acute form from *myelitis*,<sup>1</sup> especially as these are the only two acute spinal maladies beginning with fever. The pain is much more severe in meningitis, and is aggravated by movement. The contractures and cramps are characteristic of meningitis, and are not connected with uncomplicated myelitis. Hyperæsthesia, and exaggerated reflex irritability, and the lighter grade of the paresis (there rarely being paraplegia, and, if there is, it is quite late), are suggestive indications of meningitis, which should prevent any mistake. The chronic forms are of slow development, and all the symptoms increase progressively after their appearance, the paralysis being gradual and connected with contractures of the affected limbs. The paralysis may not be bilateral, as is usually the case in syphilitic meningitis, and there is rarely any extension of the disease to a higher or lower level. In meningitis there are none of the atrophic tissue changes of the myelitis, but the chronic form may so closely resemble chronic myelitis as greatly to puzzle the diagnostician. The anæsthesia that belongs to myelitis, however, is rarely present in meningitis; and, if it should be, is a late and slight symptom.

*Tetanus* may possibly be mistaken for meningitis, but such an error in diagnosis should be rare, the spasms of the former being much more general; and, besides, the temperature variations are entirely different, as the thermometric rise in tetanus is unattended by any increase in the volume of the pulse; while in acute meningitis the temperature and pulse are those of an inflammatory disease.

**Treatment.**—The acute disease must be met with energetic treatment. Local abstraction of blood by leeches or wet cups is the first indication. Rollet<sup>2</sup> has used the cautery even in the last stages, applying it from the nucha to the sacrum, and with good effect. Chauffard<sup>3</sup> has given opium in large doses in the early stages. I prefer, however, suppositories of opium or belladonna, which seem always to relieve the pain, and are attended by the additional advantage of not deranging the stomach. Blisters applied on either side of the vertebral column, iodide of potassium, and mercurials (the former in large doses, even to the amount of a drachm thrice daily, beginning, however, with a minimum dose), are excellent remedies. In chronic meningitis I have repeatedly witnessed the beneficial effects of ergot, and the notes of the case I present will enable the reader to appreciate its immediate and powerful action in a very obstinate example.

B. W., female, aged 24 years, single, domestic; admitted to hospital July, 1875.

<sup>1</sup> By the use of this term I mean not only general myelitis, but those localized forms known as adult and infantile spinal paralysis.

<sup>2</sup> Mémoires de l'Acad. Nat. de Méd., xx.

<sup>3</sup> Rev. Méd., 1842.



*July 6.* The accession of her trouble began about eight months ago, when severe pain in the lumbar region made its appearance. This was very intense, and seemed aggravated by the supine position. About ten days after this appeared, the abdomen became tender, and there were darting pains which extended about the body, radiating from the spine; this abdominal tenderness continued for two weeks, and then disappeared. She was able, at the end of a month, to "go up stairs, and to move about the house." A few weeks afterwards she noticed a loss of power in the right leg and thigh, and next in the left; and, a month later, she found it impossible to get out of bed in the morning. She said that her legs were hyperæsthetic, and spoke of feelings of "pins and needles" in the soles of both feet. She says that she thought her trouble arose from a cold that she had caught when working in a damp place. All this time her pain was quite intense, and there has been no improvement. She has great difficulty in micturition, and is constipated.

*29th.* Painted iodine on either side of the spine, and gave her gr. v. potass. iodid. t. i. d.

*Aug. 17.* Her abdomen has been distended by gas for the last two weeks. Pancreatine ʒss t. i. d., and low diet.

*24th.* This treatment has not diminished the size of abdomen. Ordered milk, rice, and beef-tea.

*30th.* Lumbar pain very severe. She can hardly move at all, and is obliged to use crutches. Injections of tr. assafetida. Charcoal and water fail to relieve the flatus. The abdominal distension is quite distressing.

*31st.* To-day another injection of the same kind did no good. Insomnia and great suffering, as the lumbar pain is severe; prefers her bed, and lies on the left side. Chloral hydrate; potass. iodide. Increased convulsive movements of legs.

*Oct. 9.* At times she has localized pain over insteps of both feet, and pain on outer aspect of right knee. For the last five days slight numbness as far up as her knees. Legs have "jerked" less for the last fortnight; can move well in bed; very slight power to move right knee; frequent desire to urinate; tympanites; some colic, pain less in lumbar region. Pulse 126, small and irritable; temperature  $101\frac{2}{10}^{\circ}$ . Blisters every other night on either side of the spinous processes.

*24th.* Abdominal pain lessened; can move legs more freely; numbness less.

*Jan. 20, 1876.* Acidi nitromuriat dil. has relieved constipation, which has been a constant symptom.

*Feb. 7.* ʒss. fl. ext. ergot t. i. d.

*19th.* Ergot has had wonderful effect. Patient left her bed yesterday, and walked to the front door of hospital (about 50 feet) and back without fatigue. She steadied herself by taking hold of the bedsteads. Has discarded her crutches.

*25th.* Walks well.

*March 15.* Goes out of hospital.

*April 1.* Discharged recovered. This patient was seen six months afterwards, and she had had no relapse.

Ergot has acted beneficially in other cases which I have treated, and I am of the opinion that it is more valuable than any other remedy in both the acute and chronic varieties of spinal meningitis. The actual cautery

applied every other day should be faithfully used, and in addition we may employ setons at the nucha or lower down. Cod-liver oil and generous diet are to be prescribed, and every measure is to be adopted that will in any way build up the patient. Should we find vertebral disease, a suitable brace, or the plaster-jacket should be provided. The advantages of Sayre's suspension treatment can hardly be overrated, and I have repeatedly seen very decided improvement follow the separation in this way of diseased vertebræ, and, consequently, removal of the pressure upon the nervous tissues.

### SPINAL TUMORS.

The growth of tumors in the spinal canal or cord is of far less frequent occurrence than in the cranial cavity and brain, but when tumors choose this locality their presence is to be much more easily diagnosed.

The varieties of spinal growths are just as numerous as those found in or about the superior part of the cerebro-spinal axis. They may be of any of the forms I have named in speaking of cerebral tumors, but those usually met with are the following:—

*Syphilomata.*

*Fibromata*, attached to the meninges, or in the substance of the cord.

*Tuberculous* (rarely).

*Myxomata.*

*Sarcomata.*

*Parasitic growths* are seldom found, and the other forms which have been spoken of in our consideration of brain-tumors are equally uncommon. *Exostoses* give rise to many obscure, but none the less interesting symptoms, while *sarcomata* are occasionally to be found attached to the inner surface of the dura mater or other meninges.

Spinal tumors are of slow growth, and of course the appearance of symptoms is consequently gradual and insidious.

**Symptoms.**—The first indications are expressions of irritation, and as a result there will be localized pain, and various disturbances of motility dependent upon the aberration of that part of the cord which is the seat of the tumor. Our knowledge of physiology of the cord will enable us to appreciate that disturbances in various parts will be followed by symptoms of pain,<sup>1</sup> hyperkinesis, akinesis, or muscular contractures expressive of involvement of the posterior, anterior, or lateral columns, but there is usually no such possible localization, as the growth generally impinges upon large tracts and works wholesale mischief. Compression is followed by still more pronounced symptoms than those attendant upon simple irritation. And there may be complete paralysis and atrophy, with muscular contractures of the members either of the upper or lower extremities. Should the tumor be situated high up in the cord, the muscles at the back of the neck may be the seat of contractures, and those of the face and neck may even suffer; if the tumor be seated lower down,

<sup>1</sup> Reynolds considers that pain in the back is more intense with carcinoma than with tubercular or other growths.

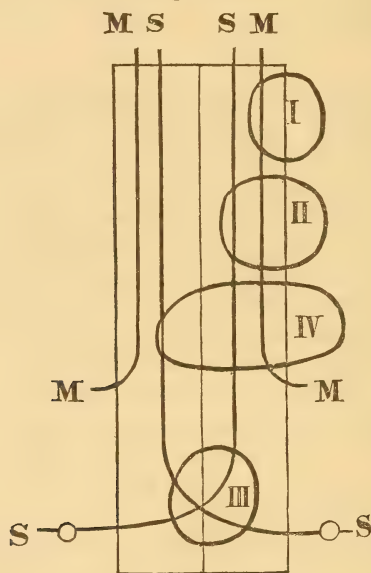
the bladder and rectum may also become involved, as in some other forms of spinal disease.

Among the early symptoms may be mentioned the constricting band which is connected with neuralgic pains that shoot down the legs. These indicate irritation of the posterior columns and nerve-roots. There is also a certain amount of painful rigidity of the spinal column. Should the anterior column and nerve-roots be subjected to the irritating presence of a tumor, the consequence of such trouble will be convulsive local spasms and increased reflex excitability. Vomiting, dizziness, and pupillary dilatation are mentioned by Jaccoud as evidences of tumor situated in the cervical region, while nystagmus and strabismus are also occasional expressions of a growth so located.

The paralysis which follows increased pressure is not always equal, one limb being more feeble than another; or there may be hyperkinesis on one side, and paresis on the other.

Unilateral irregular troubles, both of motility and sensibility, are the rule. There may be limited and well defined anæsthesia and analgesia will be found on the side opposite the lesion, while the paralysis may be the

Fig. 36.



striking symptom on the side of the tumor. This may be explained by the diagram of Radcliffe, which I have slightly modified. Supposing that Fig. 36 represents a segment of gray matter, we will consider that SS' represent sensory fibres of a nerve-root, and MM motor fibres. The sensory fibres decussate, S going to one side of the body while S' goes to the other. M and M' both leave the cord on opposite sides. A tumor, pressing upon either lateral half of the cord, such as "I," may simply paralyze motion on



the same side, while sensation remains unaffected, and both sensation and motion are intact on the other. If deeper pressure is made, supposing "II" to represent the tumor, not only would motion be paralyzed on this side, but sensation on the other. If a tumor such as "III" should impinge at the decussation of the sensory conductors, we might expect total abolition of sensation on both sides, while there would be no paralysis of motion. A tumor such as "IV" would paralyze sensation on both sides, and motion on one. When we find that there is crossed spinal paralysis, one arm perhaps being involved with the leg of the opposite side the lesion undoubtedly occurs in two points of the motor spinal track at a place above the decussation and below.

Reflex excitability is ordinarily increased in the limbs below the lesion, but it is stated that, when the inferior part of the lumbar region or the *cauda equina* are destroyed, reflex excitability is abolished after a period of six days, and that then the muscles begin to atrophy. Jaccoud<sup>1</sup> says: "There is here a new application of the law I have endeavored to make clear. As long as cerebral influence only is deficient in the inferior members, the reflex and electric motility and nutrition of muscles are intact, but when the spinal influence is in default these properties are abolished."

A case which may be detailed because of its interesting morbid appearances and which during life seemed to refute this assertion is the following, but after death an additional tumor was found higher up, which might have suspended cerebral influence, and still have left a portion of the cord capable of giving rise to reflex movements when irritated; but in some respects the case still renders what Jaccoud has said somewhat doubtful, as the question arises whether the larger tumor did not antedate the smaller, and whether the original paraplegia did not take place before the growth of the smaller tumor destroyed the cord. The patient entered the Epileptic and Paralytic Hospital September 18, 1872, and was examined by Dr. Janeway, Dr. Séguin, Dr. Mason, and myself, and the very thorough autopsy was made by Dr. Maxwell.

P. K., aged 30 years; occupation, painter; habits, intemperate. Invasion of the disease, five years ago. Relations to other diseases, disease of the spine. Seat of paralysis, lower extremities. Control of sphincters, very poor. Voluntary movements, imperfect. Sensibility, good. Speech, good. Hearing, good.

Patient denies venereal disease, and no indications of it are found on examination. He states that ten years ago, after an attack of smallpox, he noticed a pain in the lumbar region, slight and irregular in occurrence.

Accompanying this pain he has had frequent and uncontrollable desire to go to "stool," and to make water, but could not do either to his satisfaction. This all continued for about five years, when he noticed that he was gradually losing control over his lower extremities, and in five months was completely paralyzed.

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<sup>1</sup> Op. cit., p. 352.

Says the left lower extremity remained unaffected the longest, and in a short time this also became as weak as the right. Has no control over bowels, and has but little control over the bladder. Physical examination reveals a slight degree of right lateral curvature, and a marked prominence in lumbar region, and tenderness on pressure at a point corresponding to fifth lumbar vertebra. These signs seem to point to lumbar abscess, as there is slight fluctuation, and the cachexia of patient is decidedly indicative.

Both lower extremities are much atrophied, soft, and flabby. Patient very anæmic. Prescribed iron and quinine.

October 9. Patient since examined by Dr. Séguin, who says the abscess is over a point corresponding to upper third of sacrum, instead of last lumbar vertebra, as was first supposed.

14th. At the age of thirteen was struck in the small of the back with a stick. No phthisis. At beginning of trouble he had severe pains in dorsum of feet, with swelling and short lancinating pains. Pains in back part of the thighs, in loins, and about the sides of pelvis. No incontinence of feces. Curvature began about a year later than the commencement of paralysis. When limbs were extended they were agitated by clonic spasms, and increased pain in feet. As paralysis increased pain diminished, although diminution was not noticed until after contracture. In last two years no material change has taken place. Pain at irregular intervals, and occasional spasms in legs at night. Has had from the first a feeling of coldness, but never any numbness. Voluntary movements at hip-joint quite free. Knees flexible at an acute angle. Extension and flexion possible in both knee-joints to such an extent as to bring legs at right angles to thighs. No sign of voluntary movement below knee-joints. Passive movements free at hip-joints for extension, which is considerably restrained at knee-joints. Flexion free, extension beyond right angle hindered by tension of flexor muscles of thigh. More free at ankle-joints and toes; the thighs are somewhat wasted, but not truly atrophied. Left measures  $37\frac{1}{2}$  centimetres; right, 32 centimetres.

The legs show extreme atrophy, most marked on right side. Left calf measures  $23\frac{1}{2}$  centimetres; right,  $21\frac{1}{2}$  centimetres. The feet are not œdematous. The integument over lower half of tibia is apparently hypertrophied, feels elastic, does not pit on pressure; the appearance is like that of œdema. The bones do not seem to be enlarged.

When he urinates he appears to empty the bladder at once, but does it with difficulty.

*Sensibility* decidedly lessened below knee; slight impairment of feeling on posterior aspect of thighs. Sensibility much impaired below knees. Impressions of pain are perceived less acutely than normal at top of right foot; less acutely on left foot. Pricking not felt on left toes; slightly perceived on right toes.

Claims to perceive pressure of hands on both feet. *On irritating soles of feet, slight involuntary movements are caused in thigh muscles.* Legs and feet markedly cold. On left foot has ingrowing nail, with ulcerated external matrix. The right toe was seat of ingrowing nail, with ulceration, some months ago. Lower limbs perspire easily when warmed in bed. Very feeble response to faradic current on thighs; feeble reaction manifested. No response in leg muscles. Lower lumbar region presents a rounded tumor, about  $2\frac{1}{2}$  inches in diameter, projecting about an inch, and situated wholly over sacrum. The last two lumbar vertebræ are

unnaturally prominent. Moderate pressure produces no pain in tumor; several large veins lie over tumor, which is elastic to feel, and gives an obscure deep fluctuation.

Deep pressure in left iliac region produces but slight pain. The finger reaches a tumor deep in abdomen. Examination by rectum shows a relaxed sphincter; the finger meets with an apparently large promontory of sacrum, which is moderately elastic; some fluctuation. There is quite surely a tumor involving the anterior surface of sacrum. Pressure of finger upon pelvic tumor does not affect external dorsal swelling.

Patient remained in the hospital for a year after this, and finally died of exhaustion.

*Autopsy* thirty-one hours after death. Rigor mortis passing off. Abdomen of greenish discoloration. Lower extremities contracted. Left foot slightly oedematous; muscles of extensors atrophied; commencing decomposition in superficial veins; large bed-sores over sacrum.

*Brain*.—P. M. decomposition; P. M. imbibition along vessels.

*Stomach* and *Intestines* are apparently normal. The pelvic cavity was filled by a moderately firm, elastic, ovoid tumor, extending upward out of the pelvis as far as lower border of third lumbar vertebra; the psoas muscles flattened, and spread out over its upper and outer border on either side. Aorta and inferior vena cava raised and flattened by the upper end of the tumor; the external iliac vessels raised from their normal situation and course over its lateral borders. All of above-mentioned vessels empty; the ureters are over the upper border of the growth, and are tightly stretched and flattened.

*Bladder* contracted; fundus raised out of pelvic cavity; muscular trabeculae flattened; mucous membrane pale around openings of glandular follicles.

Prostate gland elongated, flattened, and atrophied from pressure.

*Rectum* raised and pressed against posterior left lateral wall of bladder. The growth had its origin behind peritoneum.

The tumor has destroyed the whole sacrum, except a small piece of its lower end, and a few small thin plates, from here and there, on the surface of its posterior attachment; the fourth and fifth lumbar vertebrae were wanting, except portions of laminae and spinous processes; the body of third has in its lower border a large concave cavity.

The tumor was also attached to the lateral wall of the pelvis; the articular surfaces of the ilia eroded; the right most destroyed. During its removal large cavities were opened, from which a thin, yellowish, viscid fluid escaped, more or less colored with blood. After removal, the tumor, with bladder, prostate, and portions of rectum, weighed five pounds; measured in long diameter twelve inches, transverse six to seven inches. In laying it open on posterior attached surface, the tumor is composed of large trabeculae and solid portions inclosing areola, which contained the fluid above mentioned.

The surface of the trabeculae was covered with small and large villi, projecting into the cysts; the general color was yellowish or yellowish-brown; in certain portions hemorrhagic. These hemorrhagic patches are softer than the yellow "consistency," and there were solid portions, where it was quite firm. Microscopic examination showed the histological structure of the tumor to be a myxo-fibroma-cavernosum.



*Spinal Cord.*—A small secondary tumor, about two inches above its lower end on left side, behind origin of anterior roots of spinal nerves. This tumor is about three-quarters of an inch by half an inch wide, ovoid, reddish, and shining, gelatinous, and attached to the "pia mater." The cauda equina has been destroyed, except a short portion of the origin of the nerves composing it; the whole cord, but especially the anterior half below cervical portion, softened, presenting numerous varicosities.

The secondary symptoms of spinal tumor are those which are generally known as "compression symptoms." All the phases of secondary degeneration follow, and after a variable time, the patient's taking off may occur from myelitis and exhaustion.

**Causes.**—The existence of the tubercular or syphilitic cachexia, the indications of former or coexisting syphilitic symptoms, and the history of the patient, may throw some light upon the spinal condition; but, after all, we know very little about the etiology of spinal or other tumors. Spinal growths are rarely found, except in adult life.

**Morbid Anatomy and Pathology.**—Syphilitic deposits are found in the spinal substance between the meninges and about the nerve-roots. The exudation resembles that found in the brain and other organs. The site of these deposits is chiefly about the circumference of the cord, and is rarely central. Tubercular deposits may affect the entire cord and its covering, but have been met with in the majority of instances in the gray matter. Jaccoud says that they are nearly always found in the gray matter of the lumbar enlargement. Tubercles may be found co-existing in the cord and brain. Myxomata are found in the cord much more often than in the brain, and are attended by separation of the nerve-fibres and great mechanical destruction. Cancerous growths may and usually do spring from the vertebræ, and are of a fungoid character. Secondary degenerations are to be found in certain cases, as well as aneurisms, organized clots, cysts, and other evidences of previous disease.

**Diagnosis.**—It is not an easy matter to distinguish the symptoms which attend spinal tumor from those of some of the other spinal diseases. We should bear in mind, however, that the indications are slowly expressed; that the paralysis is irregular; that one group of muscles may be affected at first, and then others; that the degree of lost power is not the same on both sides of the body; and, also, that perverted sensation is not the same over the two sides; that, usually, there are contractures of the limbs which need not be preceded by atrophy; and, finally, that pain is a symptom which is very constant. A diagnostic point alluded to by Leyden is that certain movements increase the spinal pain as the tumor is compressed.

**Prognosis.**—I have never witnessed a recovery from spinal tumor unless the character of the growth was syphilitic, and doubt very much whether a cure has ever been effected. It is impossible to limit the duration of disease which depends so much upon the character of the morbid

growth. Patients may last for eight or ten years; or, on the other hand, they may live a very short time, should the tumor be cancerous. Death usually occurs by pneumonia, uræmia, or some debilitating disease.

**Treatment.**—If syphilis be suspected, we are to give *very large* doses of the iodide of potassium; or, we may administer the biniodide of mercury in combination with this salt. In other states, supportive treatment or counter-irritation offers a feeble hope of relief. Morphia or muscarin may be injected hypodermically for the relief of pain.

## SPINAL HEMORRHAGE.

### MENINGEAL; CENTRAL.

**Synonyms.**—Hæmatorrhachis; hæmatemyélie (Ollivier). Spinal apoplexy.

Under this head we may consider the effusion of blood into the spaces between or under the meninges of the cord, and the effusion of blood into the substance of the cord itself.

**Symptoms.**—Very often the first intimation of the rupture is a sudden loss of power, and consequent inability of the individual to stand. It may, on the other hand, be of gradual development, the symptoms appearing in groups, one after the other. The resulting paralysis is generally complete, and the patient loses both motor power and sensibility, as well as control over the bladder and bowels, accompanied by a number of slowly-developed symptoms, with diminution of reflex excitability, although the latter may be exaggerated in some cases should the hemorrhage be small and between the meninges. The abolition of muscular power may vary in proportion to the gravity of the hemorrhage, and if it be small the patient may ultimately recover, and eventually present no indications of his loss of power. I have never seen a fatal termination before the end of several days, and doubt if such could be the case unless the hemorrhage should occur at a very high point, involving a number of the intercostal nerve-roots; but even this is improbable. Of course much depends upon the site of the ruptured vessel. If the upper part of the cord or the medulla be affected, then an immediate and fatal termination is a natural result. *Meningeal hemorrhage* is characterized by more pronounced symptoms of muscular rigidity, or by convulsions, which may be of a tetanic character. If the hemorrhage has taken place above the fourth or fifth dorsal vertebra, it is common to find obstinate priapism and intestinal disturbances, giving rise to flatus, these resulting from paralysis of the splanchnics; if it be extensive, there may be paralysis of motion and sensation from pressure exerted upon the cord, and pain and spinal tenderness are also quite marked symptoms, and in uncomplicated cases there is cutaneous hyperæsthesia. There is commonly no loss of consciousness in either variety, but when the effusion takes place in the medulla there may be conditions akin to epilepsy. In this case,

however, effusion would be very small, and the region affected would be near the circumference.

**Causes.**—Spinal hemorrhage is usually the result of a traumatism, but may proceed from various debilitating maladies and some of the zymotic diseases, smallpox playing occasionally a part in the etiology. Alcoholism, and other conditions in which the cord is congested, may predispose; or the hemorrhage may result from the rupture of an aneurism in the vertebral canal, such as occurred in Laennec's case. It very rarely takes place as a secondary accident in tetanus, so that it can be recognized before death; but at the *post-mortem* examination such pathological evidences may be occasionally observed. Traumatisms undoubtedly most frequently produce this condition; and falls, blows upon the back, or concussion following a fall upon the feet, enter into the etiology. It rarely occurs after middle age, and men are more often the victims than the other sex. It occurs in the course of myelitis, but again it may happen without any trace of inflammatory trouble to be discovered after death; and, in some instances, there is no history of injury. Such a case undoubtedly resulted from sudden congestion at the menstrual period, and is reported by Goldammer<sup>1</sup>:—

"The patient, a girl of about sixteen years, was suddenly attacked with a severe pain in her back between her shoulders, which soon passed over to her right, and after a while to her left arm. She also noticed a pain in the pit of her stomach, and found somewhat later that she could not move her right leg. Having been sent to the hospital, the examining physician found complete paraplegia, complete anæsthesia up to the mamillæ, and paralysis of the bladder, while the reflex action of the lower extremities was still intact; her temperature was normal, pulse 80; did not show any brain symptoms, but complained of pain in both arms. A few days afterwards the abdominal and dorsal muscles proved to be paralyzed, and percussion of the spinous processes of the dorsal vertebræ caused her pain. The pulse was 96; her bowels moved only when drastics were given her. A slimy discharge from her vagina was noticed. The case was considered as hemorrhage into the spinal cord below its cervical enlargement. The treatment consisted in local depletion, in the methodical use of the ointment of mercury, and in the use of drastics. The patient, having improved in general very little, died from decubitus about a year after the attack. The most noteworthy observations made on autopsy are the following: About one inch below the cervical enlargement of the spinal cord there seemed to be a compressure. A cross section through this part showed that its original diameter was reduced very much, and that the right lateral column and the adjacent parts of the anterior and posterior columns, as well as the gray substance between, were occupied by a rusty brown substance of callous consistence. The microscopic examination of this proved that it was formed of *connective tissue* inclosing fatty matter, crystals of hæmatoidine and a granulated brownish pigment; the vessels in this part had undergone fatty degeneration, their walls were thickened, and contained brown pigment; *no nervous elements* could be found in this substance; its entire length was about one tenth of

<sup>1</sup> Virchow's Archiv., Jan., 1876, and abstract in Med. News.



an inch. The adjacent parts of the medulla were *not degenerated* by softening; only a few rusty stripes and a yellowish color were noticed on their examination; the whole remaining cord was found to be intact. As no symptom speaks for myelitis as a causal element in this disease, it could only be caused by an effusion of blood into the substance of the cord: the latter probably had been provoked by suppression of the menses, for the heart and the vessels, especially those of the spinal marrow, were intact, and no injury had occurred to the patient. It is true that she stated she never had had her catamenia nor noticed any mola-mina, in spite of her age and bodily development. There were, also, no signs of menstruation noticed during her sickness. But there was revealed by autopsy the presence of a corpus luteum of the size of a pea, and certainly of long standing; and a slimy excretion from her vagina was observed a few days after the attack. These facts favor strongly the above-mentioned suggestion."

A cause alluded to by Erb is the disturbance of the balance of pressure within and without the cord. As a cause of this kind may be mentioned the sudden spinal congestion that takes place when an individual goes into a caisson or other place where compressed air is used. Dr. A. H. Smith, some years ago, alluded to a form of disease which occurred among the men at work in the caissons of the Brooklyn bridge.

**Morbid Anatomy.**—Central: hemorrhage takes place into the upper part of the cord more often than in any other locality, but the lumbar and dorsal segments may also be its seat. The gray matter is naturally more frequently the seat of hemorrhage than the white, and when preceded by myelitis or injury it will be generally more extensive than in the latter. If the hemorrhage be profuse, we will find that the cord is enlarged at the point where the escape of blood has taken place, and that it has a doughy feel. Hemorrhage into the meninges may be sometimes associated with an intracranial condition, the blood escaping from a cerebral vessel, flooding the ventricles, and passing down into the spinal cavity. Various meningeal diseases may terminate in this way, as well as spinal congestion and tetanus, and occasionally spinal tumors and vertebral disease give rise to such an effusion of blood. Old cysts have been found in the cord in some cases, but their existence is comparatively rare, and when met with they present the same appearance as is seen in the brain, though of course they are much smaller. In meningeal hemorrhage, the coverings of the cord are red and suffused, and perhaps opalescent and thickened, and there is possibly some meningitis with seropurulent collection; the effused blood may be found as a semi-organized clot, and presents, according to the time of existence, changes of color of varying depth. The size of the clot may vary from a few millimetres in diameter to a much larger size. In some instances the pia mater is torn so that there is an escape of blood into other parts. Occasionally the condition which favors the development of spinal apoplexy may lead to cerebral accidents of the same character, and evidences of such trouble may be found to coexist. Evidences of secondary myelitis are quite common about the lesion.

**Diagnosis.**—The symptoms must be distinguished from paraplegia due to myelitis, and from those of cerebral hemorrhage, which may, as Brown-Séquard has lately shown, be produced. In the former there are primary symptoms which I will discuss in speaking of myelitis, and in the latter there is usually some affection of consciousness, and some disturbance of speech. This latter variety of disease (cerebral paraplegia) is so anomalous, however, as to have but little weight as a condition to be excluded. The subsequent effects of such a hemorrhage, paralysis, contractures, etc., may be confounded with several chronic conditions. Among these are spinal tumors, adult spinal paralysis, and ataxia. The first is connected with decided hyperkinesis, is of gradual development, and is accompanied by slowly appearing symptoms. Antero-spinal paralysis or adult spinal paralysis is ushered in by fever and unattended by any loss of sensation or incontinence, and the atrophy is rapid. Locomotor ataxia is symptomatized by increased electric contractility, by *no* paralysis, by disturbance of co-ordination, by absent knee-phenomenon and by optic nerve and pupillary changes.

**Prognosis.**—If the hemorrhage takes place in the meninges or in the lower part of cord, the prognosis is perhaps better than if its seat is in the cervical or dorsal segments. In the first instance the patient may live some time or ultimately recover, but in the latter the probability of sudden or early death is almost certain. Grisolle<sup>1</sup> says "Spinal hemorrhage runs a rapid course. A single patient has survived forty days; the majority, however, succumb at the end of several days, by suspension of respiration. Among others death is hastened or produced by the development of bedsores. Nevertheless, spinal hemorrhage is not necessarily a fatal condition." He refers to a case observed by Cruveilhier, and states that this is the only cure of which he has known. Erichsen,<sup>2</sup> however, has reported recoveries which have taken place in cases which were of traumatic origin; so the prognosis is perhaps not so bad, after all.

**Treatment.**—The early treatment of spinal hemorrhage should consist of cold applications to the spine, perfect quiet, and rest. Subsequently ergot and belladonna will be of great benefit. The former may be injected hypodermically in the form of its extract, rather free doses being used which should be repeated frequently. Five or even ten grains may be used. Iodide of potassium in full doses does good sometimes. Blistering and leeches to the painful point in the back are next in order, and later on the actual cautery is the most serviceable external agent.

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<sup>1</sup> Grisolle, *Path. Interne*, vol. i. p. 659.

<sup>2</sup> On Concussion of the Spine, etc.

## CHAPTER VIII.

## DISEASES OF THE SPINAL CORD.

## SPINAL HYPERÆMIA.

## (A) SPINAL CONGESTION; (B) SUBACUTE SPINAL HYPERÆMIA.

Two varieties of spinal hyperæmia exist: one of sudden origin, and of a sthenic character, which I prefer to call *Spinal Congestion*; the other of slow progress as compared to the first, and characterized by *accumulation* rather than congestion, which I will speak of as *Subacute Spinal Hyperæmia*.

## SPINAL CONGESTION.

This first variety, which has been excellently described by C. B. Radcliffe,<sup>1</sup> is not so common as the latter, or at least such has been my experience. It is apparently a serious condition, and may somewhat puzzle the incautious observer who may mistake it for some one of the organic diseases; but it has certain distinct features which do not belong to the organic neuroses, and I think there should be no difficulty in making a diagnosis.

**Symptoms.**—The following may be the symptoms of an attack of *Spinal Congestion*. The patient probably attracts the notice of his friends by telling them that he cannot get out of bed, that “he feels as if he were a lump of lead,” or that his “legs and arms are made of wood.” He cannot move, and complains repeatedly of his utter weakness; he sighs, and may complain that the room is close, and ask to have a window opened; he is able to appreciate any warm substances that may be applied to the surface, and very acutely feels pinching or the prick of a pin. The legs, he says, seem very cold, and he requires extra covering; he has backache and pains, which run down the back of the thighs, but pressure does not aggravate the pain in the back, which is only relieved by lying upon the side or belly. His mind is clear, but he is restless, suffers for want of sleep, and is extremely uncomfortable. The functions of the bowels are perhaps interfered with, there being constipation; but there is never incontinence of urine or feces. The patient becomes paralyzed, and such paralysis is rather sudden, and may take place during the night, or perhaps more gradually after the appearance of pain and the other symptoms just mentioned. Reflex action is abolished, and electro-muscular contractility is increased.

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<sup>1</sup> Article in Reynolds's System of Medicine, American edition, vol. i., p. 942.



Radcliffe calls attention to the wasting of the muscles, but I have never seen more than the general atrophy which would occur from disuse of the lower extremities, for the patient may sometimes lie in bed for months before he regains the lost power. The duration of the attack rarely exceeds six weeks, but there is a possibility of a second attack. The paralysis is generally paraplegic, though it may be irregular in its onset, one leg or arm being affected before the other, and in some cases it is general. The spinal pain seems to be increased by warmth, and the patient will feel the ice-bag to be very grateful after lying upon his back for a long time on a warm bed. These pains are as a rule unaffected by movement, which is not the case in meningitis. Bed-sores as a feature of the disease are never seen, and for this reason no suspicion of transverse myelitis should arise.

#### SUBACUTE SPINAL HYPERÆMIA.

**Symptoms.**—The expressions of this condition are very slowly manifested, and are very often mistaken for those of the opposite condition—anæmia of the cord. Tingling and heaviness of the limbs may distress the patient, and render him disinclined to take exercise or remain standing for any length of time, and much of his want of energy may be mistaken for laziness. These symptoms are especially disagreeable towards night in those who have walked much during the day, and there is an uneasy, tired feeling, which is only relieved by change of position; and the patient seeks in vain for a comfortable place to rest his weary limbs, and only finds it when he lies upon his bed or sofa. There may be cutaneous anæsthesia, and occasionally hyperæsthesia, but these sensory troubles are by no means common. There may also be the “constricting band,” which is so usually suggestive of inflammation, and there are vague undefined pains in the thighs, legs, and back, which are extremely distressing. The temperature is lowered, and there may be the same oppressed breathing which is such a marked feature of the acute variety. Decided paresis is rare, and, if it should take place, it is nearly always paraplegiform, and not general, as it may occasionally be in the acute variety. Should this be the case, we will find the same impaired condition of reflex excitability and normal electro-muscular contractility which characterizes the more active variety of spinal hyperæmia. The tendency of the disease is to disappear under proper treatment, and in its worst forms is neither a grave nor lasting trouble, and should not be looked upon with alarm.

**Causes.**—Women seem to be more subject to the first form than men, and this is probably owing to irregularities of the menstrual condition. Uterine conditions, symptomatized by dysmenorrhœa or amenorrhœa, may be, and often are, its sole causes. Among men, the long continuance of the erect position seems to favor the gravitation of blood, and hypostatic hyperæmia of the spine is thereby induced. A few years ago I satisfied myself that the maintenance of the erect posture for a long-continued period resulted in a great deal of mischief. My investigations were chiefly

among car-drivers, who were compelled to stand upon the platform of the city railroad cars for a period of from fourteen to sixteen hours daily. Spinal congestion, varicose veins, and other vascular changes were common and serious results; and the spinal troubles were only relieved by a long rest. Venery, alcoholic intemperance, and malaria are often causes of spinal hyperæmia; and suppression of any bloody discharge, such as the menses, or that from hæmorrhoids, will be apt to be followed by more or less spinal hyperæmia. Among the more serious causes of spinal hyperæmia may be mentioned the fevers. The spinal congestions which usher in some of the exanthemata are symptomatized by back pains, etc., and do not properly come under this head for discussion; but there are conditions which play a most important part in the etiology of spinal congestion. The malarial cachexia very frequently induces a condition of spinal hyperæmia which misleads the observer, and the true cause may be lost sight of under the periodic character of the painful exacerbations. This we should take into account if there be any suspicion of malarial poisoning. I have seen many cases of very decided subacute spinal hyperæmia which followed intermittent fever. The disease had become masked to some degree, so that no chill was complained of; but the individual suffered more at some parts of the day than at others, and, in one case of this kind, there was some loss of power, which was increased daily at a certain hour, and never seemed to disappear entirely.

**Morbid Anatomy and Pathology.**—What I have said in speaking of cerebral hyperæmia may be referred to in explanation of the appearances met with in spinal congestion. The gray matter will be found to be quite dark, and the vessels are usually enlarged. The white matter is often of a pinkish hue, and there may be areas of hyperæmia which are localized; or the suffusion may be general. Microscopically examined, the cord will be found to have undergone very slight changes, and they may consist only in increased vascularity, enlargement of capillaries, and perhaps some exudation beneath the vascular sheaths. The vessels of the meninges are engorged, and there are to be observed small ecchymosed spots, or occasionally an effusion of serum. The symptoms of the disease result from pressure upon, and irritation of, the nervous elements; and the violence will depend upon the site of the most decided hyperæmia. The gray substance, when subject to pressure from distended vessels, gives rise to the pain in the back, and cutaneous hyperæsthesia, as well as the spasmodic movements which symptomatize the aggravated forms. Spinal hyperæmia is directly induced by blood defects and disease of other organs, and it is favored by the anatomical structure of the parts concerned. The tortuous course of the veins, and the absence of valves, are, according to Jaccoud, among the latter. The stasis of blood in their interior, which follows forced respiration, such as must be caused by violent exertion, or by disease of the thoracic and abdominal organs which to some degree arrests the return of venous blood from the cord, favors hyperæmia.

**Diagnosis.**—Spinal meningitis, myelitis, and spinal irritation are the diseases with which it may be confounded.

1st. The spinal pains of *meningitis* are increased, as has been shown, by movement, which is not the case in spinal congestion, and there is a muscular rigidity in the first-mentioned disease which does not exist in this.

2d. *Myelitis* differs from spinal congestion for the reason that complete anæsthesia, wasting, loss of electric contractility and sensibility, reflex-excitability, incontinence of urine and feces, and bedsores, belong to the former.

3d. *Spinal irritation* (anæmia?). The spinal tenderness is increased by pressure in anæmia, and there is no cutaneous tingling. There are troubles of other organs, and generally a variable amount of hysteria.

**Prognosis.**—The chances for recovery are very good, provided active measures are at once taken to reduce the fulness of the spinal vessels. If the condition becomes a chronic one, even then much may be done to improve the abnormal state of the circulation. In many cases, however, it precedes myelitis, particularly when it takes the slow course which I have described as subacute spinal hyperæmia, or it may lead to atrophy; but this tissue-change is more directly induced by spinal anæmia.

**Treatment.**—The local application of cups, counter-irritants, and cold may all be practised; and, in addition, we may use either hydrobromic acid, the bromides, or ergot, in full doses; or belladonna till some of the toxic effects are produced. It is never well to prescribe alcohol, strychnine, or iron in these cases, or any agents which increase central irritability, and I have witnessed disastrous effects from their use. The Turkish bath is, I think, one of the best adjuvants to these forms of treatment. As a local application to the spine, I have directed the patient to procure a strip of adhesive plaster, which should extend from the lower cervical vertebra to the sacrum. This is to be warmed and dusted with red pepper, and then applied to the back. It is a very excellent form of counter-irritant, and may be worn for some time. The cups may be wet or dry, according to the severity of the case, although I prefer the former. Should there be any pronounced symptoms, these are to be used two or three times a week. It must be borne in mind that general treatment, such as the re-establishment of fluxes which have been interrupted, and the regulation of the functions of the excretory organs, is to be undertaken as early as possible; for, like cerebral hyperæmia, the condition is nearly always one that is secondary. As an immediate remedy, one of Chapman's bags may be filled with ice-water and applied to the back for ten or fifteen minutes at a time, or the ether spray will answer the same purpose.



## SPINAL IRRITATION.

(SPINAL ANÆMIA ?)

**Synonyms.**—Ischémie de la moelle. Anæmie de la moelle.

The brothers Griffin<sup>1</sup> were the first to describe this interesting affection, and since the appearance of their first paper in the *London Medical and Physical Journal* in 1829, very little has been added to our knowledge of this condition, which was fully considered so many years ago. The pathology of the affection was by the Griffins supposed to consist primarily in an irritation of the sympathetic ganglia, and they divided their cases into three varieties, viz., those in which the cervical, dorsal, or lumbar portions of the sympathetic nerves were involved. In later years other observers, consider the affection due to an anæmic condition of the cord, and go so far as to attempt to localize anæmia of the different columns, I am disinclined to agree with them, not only because I believe that spinal irritation depends sometimes upon hyperæmia, but I think that this condition is due more to a loss or abnormality of cell-functions. I am therefore disposed to adopt the views of the Griffins, and consider "spinal irritation" to be a condition due to a primary perversion of the functions of the sympathetic system, or to a secondary ischæmic state, and that in some parts of the cord both abnormalities of circulation exist. Dr. V. P. Gibney advanced the view before the American Neurological Association (session of 1877) that spinal irritation was, in the majority of cases, a meningeal affection, and was usually the result of injury of some kind. In support of this theory he brought forward a number of cases, all of them of great interest. I am strongly inclined to accept Dr. Gibney's explanation, but not in its entirety. Spinal irritation is very probably due not only to affections of the cord alone, but to the meninges as well, as the symptoms of spinal tenderness suggest. That a great many cases arise from disordered functions of other organs, there can be no doubt, and the history of injury is very often absent.

**Symptoms.**—The indications of spinal irritation are quite varied, but there are several which are distinctly pathognomonic. One of these is spinal tenderness. If the observer makes firm pressure with his thumb at different points over the intervertebral spaces, he may cause the patient to wince where a painful point receives the pressure. These tender spots may be either in the cervical, dorsal, or lumbar regions, but more often the cervical or dorsal. Sometimes the skin is so hyperæsthetic at these places that the pressure of the clothing is sufficient to cause the wearer great discomfort; and such patients, be they women, are fidgety and irritable. Pressure made at certain points may be followed by pain, not only in the region pressed upon, but at distant parts; for instance, in one of Griffin's cases pressure made over the dorsal vertebra was followed by

<sup>1</sup> Observations on Functional Affections of the Spinal Cord and Ganglionic System of Nerves, etc., by Wm. and Daniel Griffin. London, 1843.

pain in the sternum. Pain also of a darting or lancinating character follows such pressure, and sometimes when the lumbar region is its seat there may be twinges which travel down the crural and sciatic nerves. So, too, may there be radiation of pain about the chest when the dorsal portion of the cord is subjected to this procedure. Pressure over the cervical intervertebral spaces produces vertigo, headache, and nausea. With irritation of the cervical region, vertigo is quite pronounced. Memory is affected, and hysterical manifestations are quite common; while insomnia and headache, disordered vision and facial neuralgia, vomiting, and respiratory troubles are all prominent symptoms. The headache is connected with soreness of the scalp, and is of a neuralgic character, and the fifth nerve is so extensively affected that toothache, faceache, and deep orbital pains when they occur, are almost intolerable. As an evidence of disordered function of the fifth nerve, there may be trophic changes in the cornea, such as ulceration, and there is in some cases keratitis. Cervicobrachial neuralgia may exist in addition to the facial neuralgia, and may be either one-sided or bilateral, and pressure made upon the cervical vertebræ may greatly aggravate the neuralgia. Diplopia, amaurosis, and other visual troubles are annoying in the extreme, and the intense hyperæsthetic state of the organs of special sense may give rise to hallucinations of sight or hearing. There is not rarely photophobia of a distressing character, so that the individual is obliged to stay in a darkened room. Deafness is an occasional symptom, and ringing in the ears is an indication of cerebral anæmia co-existent with the spinal troubles. The gastric mucous membrane may be in an extremely irritable condition, so that the food is speedily ejected, and with the vomiting there is nausea with vertigo. The spinal origin of this symptom may be satisfactorily proved by applying a blister to the painful spot. Various respiratory and cardiac irregularities are quite constant accompaniments of spinal irritation. Among these are attacks of dyspepsia, angina, palpitation, coughing, or a sense of pressure and discomfort in breathing, asthma, etc. Urinary troubles may exist when the morbid spinal condition is situated lower down, and often ovarian neuralgia. Convulsive movements of the legs and obstinate constipation swell the list of symptoms. A form of paraplegia, usually of an hysterical nature, but sometimes so constant as to seem to be dependent upon some organic lesion, occasionally symptomatizes the disease. There is even lowered temperature, though the patient may complain of subjective sensations of warmth; but the paraplegia is never attended by any evidences of the real condition which follows myelitis. The action of the bladder and rectum is normal, and the electro-muscular contractility and reflex excitability are, if anything, increased, and the anæsthesia or hyperæsthesia, if it exists, is quite unimportant.

The following history was given to me in the patient's words, and is so graphic that I consider it worthy of reproduction:—

*1st year, 1867.* There was some cerebral anæmia. Inability to think consecutively, or to do anything that required looking after; constant

nausea and dizziness; a burning in head and spine, and an occasional deep seated and momentary pain in the head; an excessive demand for pure air; extreme hyperæsthesia of skin; sleeplessness; worried feeling in the ovaries.

*2d year, 1868.* Head symptoms slightly improved; body grew weak and tremulous; felt as if starving to death, though with good appetite for nourishing food. Nausea not constant, but occurring every night between nine and ten, and lasting about an hour.

*3d year, 1869.* Mind grew painfully active, it was impossible to stop thinking, asleep or awake; gradual loss of use of arms and legs, with distressing jerkings of latter; hysterical; light and sound almost intolerable.

*4th year, 1870.* Commenced walking after lying in bed seven months. Dizziness, sleeplessness, tremor; burning in head and spine continued.

*5th year, 1871.* Same as fourth year, with some alleviation.

*6th year, 1872.* Material changes were more sleep, arrested condition of brain, and tremor not constant.

*7th year, 1873.* Dizziness, which had been constant from the beginning, ceased. Ability to converse, and listen to any amount of reading, attend lectures, etc. Pain or distressed feeling in head most of time. More depression of spirits than ever; sleep full of nightmare. Neuralgic pain; appetite indifferent; bowels torpid; menses irregular and overabundant, extremely painful, and prostrating.

The patient was 29 years old, and married. She is in appearance anæmic, evidently of a strumous diathesis, and somewhat hysterical. Her pupils are dilated, and there is decided muscular asthenia. She cannot read, and, when she attempts to do so, there is a peculiar dizziness, or, as she very pertinently calls it, a "nausea of the brain." If reading is persisted in, the dizziness is excessive, and there is ultimately vomiting. Her headache is vertical, and some uneasiness is produced by pressure made over cervical vertebræ. Her urine is copious and abundant, and contains phosphates. Constipation is persistent and obstinate. At my request Dr. Loring examined her eyes with the ophthalmoscope, and found atrophy of the left optic disk.

*Jan. 30, 1874.* Strychnia, iron, and phosphoric acid were given, and absolute rest required and enjoined; and one month later she returned, feeling very much improved. It is possible for her to read two hours at a time without being fatigued, and her spirits are very much improved; her depression has somewhat disappeared, and she sleeps much better. A curious feature of this woman's disease was excessive somnolency during the day, and it was often necessary to use violent measures to arouse her from her very profound sleep. During the evening she became very animated and bright, talking brilliantly upon all subjects, and it was not until midnight before she again felt a disposition to sleep. In her case evidently the menorrhagia was the cause of the anæmia.

**Causes.**—The victims of spinal irritation are nearly always women, and very rarely men. It may safely be said that nine-tenths of all the cases are females. It rarely occurs before puberty, but after that time may make its appearance, and then is generally dependent upon, or associated with, irregular or profuse menstruation. It not rarely begins at the menopause, but is more often of earlier origin. Hereditary predisposition seems to have much to do with its development, and various mental causes



play an important part in its production; care, worry, and overwork being among these. Various debilitating diseases, childbirth, and bad habits, may be enumerated as additional causes.

**Morbid Anatomy and Pathology.**—Spinal irritation being a functional disease, it is impossible to find any *post-mortem* indications, unless they, perhaps, are foci of low inflammatory action, such as thickening of the neuroglia, or simple atrophy.

As to its pathology, I have already expressed my views in regard to the probability of both hyperæmic and anæmic conditions as pathological factors. It is impossible, I am convinced, to locate the point of irritation in either of the columns, and any attempt to do so is an impossible refinement of diagnosis. We may approximate its seat by the region of tenderness, and the predominance of special groups of symptoms; and this is all that I believe to be possible. Spinal irritation may undoubtedly result from—1, reflected irritation; 2, impoverished blood-supply; 3, local changes dependent upon disease of adjacent tissues.

The labors of Brown-Séquard, Bérnard, and lately Lauder Brunton, have showed satisfactorily the intimate relation between the sympathetic and cerebro-spinal systems; and the observations of the former are especially valuable because of their pathological bearing. Not only may distant organs send irritating impressions to the cord, to be followed by vaso-motor stimulation, contraction, and subsequent relaxation of the vessels, but the intra-spinal circulation of impure blood may produce local irritation, imperfect nutrition of the nerve-cells, shrinkage of the nervous tissue, and œdema of the perivascular spaces. The chain of inhibitory ganglia, described in such a beautiful manner by Brunton, places in close relation the different parts of the cerebro-spinal axis, so that there is nearly always a disturbance of several organs when the harmony is affected.

The vascular cramp of Nothnagel will account for various ischæmic conditions in certain parts, while circulation in neighboring districts may be perfectly normal. Bidder<sup>1</sup> has also shown that complete alteration of vascular calibre is impossible, so that at best there is contraction but at a certain point, while the other part of the vessel may be dilated.

Bidder's experiments also demonstrated that *excitement* or exaggeration of function may exist with depressed function at the same time, in a compound organ.

It is therefore reasonable enough to consider that spinal irritation is not altogether dependent upon spinal anæmia.

The production of special symptoms is explained by the involvement of sympathetic, cranial, or spinal nerve-roots. The headache may result from cerebral anæmia, as may also the mental and hysterical symptoms; while the visceral disturbances arise from sympathetic derangement of the abdominal organs. The pain resulting from pressure is due to im-

<sup>1</sup> Die Reflexe eines der sensiblen Nerven des Herzens auf die motorische des Blutgefäßes.

pressions conducted to the over-sensitive centre by the cutaneous nerves. It is almost unnecessary to allude to the production of spasms, reflected pain, and the numerous dysæsthesia.

**Diagnosis.**—Spinal congestion, spinal meningitis, and incipient inflammation of the cord may suggest themselves to the observer. As to the first, differential diagnosis is often impossible, unless there be actual paresis. The absence of great spinal tenderness is also an element in diagnosis. Spinal meningitis is connected with tenderness, but it is not aggravated so much by pressure as by muscular movements. There are also present muscular spasms of a painful character.

Myelitis in the beginning is attended by waist constriction, which is too marked to be mistaken; and besides paralysis of motion and sensation, there is atrophy, as well as progressive symptoms. The presence of gastric disorders, which are so marked in nearly all cases of spinal irritation; of headache, and great languor, a generally depraved physical state, and the existence of uterine trouble, should all be taken into account.

Griffin alluded to several other disorders likely to produce some of the symptoms of spinal irritation. These are rheumatism, which is sometimes causative of spinal soreness, and various acute diseases, which, however, present so many symptoms of a distinct character as to do away with any chance for mistake in diagnosis. The pain of rheumatism is generally so severe and absorbing that the patient's mind is constantly directed to it, while affections of the joint usually coexist.

**Prognosis and Treatment.**—If the patient be promptly taken in hand it is often possible to cure the disease, but I am inclined to consider well-established spinal irritation the most discouraging and intractable functional neurosis that is to be met with. Commonly connected with ovarian or uterine derangement, it defies the best-directed efforts of the physician; and, if the factor cannot be removed, the patient becomes a confirmed invalid. It is, therefore, proper in all cases to search for the cause, and in three-quarters of the female cases it will be found in the pelvis. If there be general anæmia, or some other depraved condition of the system, we are to "build up" our patient with cod-liver oil and tonics, and a very excellent one is the following:—

R. Ferri et ammon citratis, ℥iij.;

Tr. gentianæ, ℥iv.—M.

Sig.—A teaspoonful in water after eating.

Phosphorus, either in the form of Thompson's solution, or the phosphuretted oil, quinine, pyrophosphate of iron, Horsford's acid phosphates, the syrup of the combined phosphates, are all in order. Nutritious food and extract of malt are to be given, and a liberal use of stimulants is strongly recommended. Strychnine sometimes does good, and at others a great deal of harm; and in cases where there is very severe pain, I prefer other remedies.

Opium in small doses is often of great value, and its effects are immediate and excellent. External counter-irritation, either by the actual cautery applied on the painful points, a blister, or some irritating ointment, is advised, and if vomiting be present, a blister on the epigastrium, subsequently dusted with morphia, allays the irritability of the stomach. I have used with success, and would recommend, galvanism (the descending current), the positive pole being placed upon the nucha, and the negative in the groin. Applications lasting five or ten minutes every day, or every other day, are sufficient.

Galvanization of the cervical sympathetic is an important form of auxiliary treatment. Heat and cold alternately applied to the spine are followed by excellent results; or Chapman's ice-bags, filled with hot water, and placed in contact with the spine for fifteen or twenty minutes daily, are beneficial.

Open-air exercise, Turkish baths, and massage, all help the patient; and Mitchell's rest-treatment, already described, is one of our best modes of treatment in confirmed cases.



## CHAPTER IX.

## DISEASES OF THE SPINAL CORD (CONTINUED)

## INFLAMMATION OF THE SPINAL CORD—MYELITIS.

**Synonyms.**—Myelitis. Myélite aiguë, chronique. Rückenmarkentzündung.

**Definition.**—Inflammation of the spinal cord, usually attended by paralysis of motion and sensation below the seat of the spinal lesion, by involuntary stools and incontinence of urine, and by absence of reflex excitability and electric contractility in the paralyzed parts, and a tendency to extension upwards, results in death in a very short time from paralysis of the intercostal muscles, especially should the pathological condition be an acute one. Inflammation of the spinal cord may extend across the cord, when it is called *transverse myelitis*; or longitudinally, when the terms *ascending* or *descending* are applied. The features of an attack of transverse myelitis, which, as an acute condition, is so rapid in development that it suspends the functions to a great extent of the columns of the cord, so that we get a simultaneous or rapid impairment of the conductors of motion and sensation, and the disordered functions of organs innervated by nerves coming from the cord below the level of the diseased portion; or, on the other hand, the integrity of the different conductors of the cord may be gradually impaired, so that many months or years may elapse before the morbid process extends across a plane, destroying successive parts. In the other forms in which the inflammatory process travels upwards or downwards, the loss of function is more irregular. Still another form exists, in which the periphery is affected, with or without meningeal complication.

## ACUTE MYELITIS.

**Symptoms.**—The disease begins rather suddenly, generally with pain in the back, which is aggravated by pressure, and an uneasy sense of tightness about the body. These unpleasant sensations may be preceded by formication and tingling of the feet, some loss of power, and the development of more or less fever, during which the temperature may be very much elevated. This is especially the case when the upper part of the cord is involved. These symptoms are followed in several hours, or after a day or two, by loss of power in the lower limbs and by an aggravation of the spinal pain. The patient will find it impossible to pass his urine, and if he is not relieved by a catheter will suffer great distress; or

there may be final relaxation of the sphincter, and it may flow from him without his knowledge. These symptoms are sometimes presented before a physician is called in, and at his visit there may be complete paralysis of the lower extremities. The surface of the limbs is cold and utterly devoid of sensation, and the soles may be tickled or the muscles pinched without any attempt being made upon the part of the patient to withdraw his feet. This reflex excitability, however, is not always lost in the beginning, but may be present when the onset of the disease is gradual, and the patient is entirely unconscious of the occurrence of these movements. If a heated substance be applied to the back, it will be found that its presence will not be appreciated below the point of spinal inflammation, but when it is passed over the diseased tract the pain is greatly increased. Above this level, normal sensibility exists, and the degree of heat is readily perceived. The attention of the physician is attracted by the ammoniacal odor of the urine, which, as has been stated, may flow from the patient without his knowledge, and the contents of his rectum may pass away in the same manner. Hyperæsthesia is an exceptional late feature, but it may form one of the initial symptoms in conjunction with trembling of the limbs. After the paralysis takes place, the temperature is lowered several degrees, and circulation is very defective. At the end of a week there may be indications of the upward extension of the spinal inflammation if it be progressive, and it is sometimes recognized by the tendency to priapism and the distress in breathing, and with these there may be hiccough and hurried respiration, the number perhaps reaching 48 in the minute. Bedsores form over the sacrum, and there is every appearance of approaching dissolution. The skin becomes clammy, and there may be rigors; while the pulse grows small, fluttering, and the voice very weak, and ultimately the patient dies, his mind remaining clear to the end. If, however, the structural alteration progresses upward, it is very probable that the mode of death will be asphyxia. As exceptional instances, cases have been recorded in which there was myelitis of the upper part of the cord, with complete paralysis of the upper extremities, while the lower limbs, the bladder, and rectum were not affected, and other equally rare forms are occasionally noted. When the dorsal portion of the cord is the seat of inflammatory action, the respiratory symptoms are immediate, and the breathing becomes embarrassed at once. The pneumonia occurring so often in a late symptom of myelitis is undoubtedly of nervous origin, and commonly indicates the implication of the medulla. The pneumonia is complicated by some bronchial trouble. Vulpian<sup>1</sup> and Arnozan<sup>2</sup> lately have given consideration to the connection between spinal and pulmonary diseases, under certain circumstances, and the former is of the opinion that the sympathetic roots of the intercostal nerves are involved.

The prominent symptoms of this interesting disease may be summed up as—

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<sup>1</sup> *Maladies de la Moelle*, p. 185. <sup>2</sup> *Des lésions trophiques, etc.*, p. 198. Paris, 1880.

1. *Paraplegia* of sudden or gradual origin, attended by anæsthesia and analgesia, but usually preceded by dysæsthesia of various kinds, or actual hyperæsthesia. It may be accompanied in the beginning, according to Radcliffe,<sup>1</sup> who has observed this symptom in severe cases, by "uncontrollable restlessness." Paraplegia is nearly always the form of lost power, though in rare cases there is hemiplegia. There may be, in exceptional cases, variations in sensibility, the symptoms of anæsthesia being absent when the anterior columns are alone partially affected. Again, in other cases one leg may be paretic and the other anæsthetic. The onset of the paraplegia may be very sudden, and the disease prove rapidly fatal. Jaccoud<sup>2</sup> has seen one case in which the paraplegia developed in thirty-six hours from the commencement of the disease. *Eighteen hours* afterwards, the autopsy revealed a purulent meningo-myelitis of the entire lumbar and part of the dorsal segments of the cord. The extent of the paraplegia is of course governed by the seat and course of the myelitis. If the lumbar portion of the cord be destroyed, the lower extremities, and the muscles of the abdomen and sphincters will be paralyzed; if the myelitis extends so that the dorsal portion and the *cilio-spinal* centre are involved, the arms are paralyzed, and pupillary changes with irregularity of cardiac functions are produced. When the lesion is still higher, and the cervical portion of the cord is involved, there may be, in addition to all these forms of paralysis, various difficulties in swallowing, speech, and respiration, and the patient dies from asphyxia.

2. *Reflex excitability* is generally abolished entirely, or impaired to a great extent. Occasional exaggeration is seen in the earliest stages, or when the myelitis involves limited regions, especially the lumbar segment. Jaccoud says: "Durant la période d'exagération (hyperkinésie réflexe) le segment lombaire soustrait à l'influence du cerveau manifestait son action proper avec la puissance accrue qu'elle tirait de son isolement; durant la période d'abolition (akinésie réflexe) cette action propre ou spinale est anéantie parceque les éléments qui en sont doués sont détruits."

3. *Electric contractility and sensibility* are abolished or greatly lowered. The only exception to this rule is when the reflex excitability is increased.

4. *Muscular atrophy* as a result of severance of spinal innervation sometimes follows. This may take place in from four to six weeks. The atrophy is general, and is of course attended by absence of electro-muscular contractility and by coldness of the surface.

5. *Bedsore*s and other evidences of defective cutaneous innervation are present. The skin becomes swollen, or there may be at first great dryness and redness, or œdema at the points subjected to pressure. A hard, red bullous nodule may form, and subsequently break down, and sometimes large patches of tissue are rapidly destroyed. According to Ash-

<sup>1</sup> Op. cit., p. 314.

<sup>2</sup> Path. Interne, vol. i. p. 315.



urist bedsores are more frequent when the lesions of the cord are low down.

In hemiparaplegia when the lesion is unilateral the bed sore is also unilateral and upon the side opposite the lesion, and bearing in mind the law of Brown-Séquard, loss of power and vaso-motor paresis with hyperæsthesia upon the side of the lesion and anæsthesia on the opposite side, the bed sore appears on the anæsthetic side.

Arnozan reports a case in which a monoplegia affecting one limb was followed by bedsores upon both buttocks, that upon the paralyzed side being one and a half centimetre in its largest diameter, while that upon the other was the size of a silver dollar. The paralysis was at first supposed to be cerebral in origin, but the occurrence of violent lumbar pain and atrophy supported its spinal character. Cases are on record where a brisk arthritis developed upon the sound extremity, while upon the other a bed sore appeared.

Brown-Séquard according to Arnozan believes that the occurrence of bedsores is most frequent in cases where there is incontinence of urine.

6. *The sphincters are paralyzed*, the urine is intensely alkaline, the walls of the bladder being paralyzed, and as a consequence a certain amount of urine remains in that organ in a decomposed state, and rapidly induces an alkaline reaction in that which may collect in addition before it is discharged. Brown-Séquard is inclined to consider that this condition of affairs is pathognomonic of disease of the dorsal region, and I infer holds that it is essentially a nervous symptom. Leroy d'Etiolles<sup>1</sup> has alluded to cases of paraplegia, the so-called *paraplégie urinaires* which follow bladder troubles in which cystitis with purulent and decomposed urine, and perhaps ulcerated thickening and local paralysis of the vesical walls are found. Frequent catheterization or sounding aggravates the trouble, and a myelitis may result either as a reflex nervous trouble, or as a result of absorption. Radcliffe alludes to a reflex spasm of the sphincter ani which occasionally occurs in this disease, but this symptom is so exceptional as to need but passing comment. The paralysis of this muscle is ordinarily so complete as to be followed by the almost constant escape of softened feces and watery discharges. The sphincter ani sometimes however shows an abnormal amount of reflex excitement. A favorite subject with those who endeavor in courts of law to prove spinal disease and obtain heavy damages, is the possible atrophy of the male parts of generation. Such a consequence of myelitis is exceedingly rare, though Curling has admitted that wasting of the testicles may follow. Arnozan quotes Klebs, who says that often when wounds of the lumbar cord are near the genito-spinal region, or in connection with certain paraplegias the spermatozoa disappear and there is cellular degeneration.

7. *Increase of temperature and pulse* call for no special mention. Occurring with paralysis of the lower extremities and no loss of consciousness they can symptomatize but two acute spinal affections, myelitis and

<sup>1</sup> Des Paraplégies, 1856.

meningitis. The spasmodic movements of the latter disease, however, are not observed in myelitis, so that it possesses at least some diagnostic importance. The temperature varies from the normal standard to  $104^{\circ}$  or  $105^{\circ}$ , and the pulse may reach 160.

8. *The constricting band* sensation or paræsthesia, which is more marked in myelitis than any other form of spinal disease, is generally likened by the patient to that which might result if a tight cord were tied about the body. It is usually located at the waist, and sometimes when it is not complained of may be developed by a sharp blow on the back, or by the application of an electrode to the spine.

#### CHRONIC MYELITIS.

**Symptoms.**—The disease sometimes takes a more slow course. The paralytic symptoms are much less sudden in their onset, and occur one after another, so that the extension of the inflammation may be sometimes traced. For some time, perhaps for several months, there may be disorders of sensation, such as tingling spinal pain, and the “constricting band.” The perception of pain in the affected limbs, though not entirely abolished, is greatly impaired.

Charcot,<sup>1</sup> Romberg,<sup>2</sup> and Cruveilhier<sup>3</sup> have called attention to the curious mistakes sometimes made by patients in locating painful sensations. Pain following the pinching of one leg is referred to the other, and the painful impression may take several seconds to reach the sensorium. In one of Romberg’s patients pressure upon the toe was referred to the hip. Cruveilhier’s experiments demonstrated that an interval of from fifteen to thirty seconds elapsed sometimes before any sensation was excited, and that the impression had to be made several times before it was perceived. Electric contractility is perhaps increased, and reflex excitability is very much exaggerated, and may be followed by very violent movements. Thus, when a warm bottle is sometimes applied to the feet, though the temperature is not so high as to cause discomfort to a healthy person who touches it, the patient’s legs will be violently drawn up; this always suggests a meningeal complication. Dysæsthesiæ are referred to, and pains in the joints and bones, especially aggravated by humidity of the atmosphere, are spoken of by the patient. The paralysis of motion is much less extensive than it is in the acute form and in the beginning; and spasms of the muscles of the lower extremity are quite violent. Subsequently, however, they disappear as the loss of power becomes more complete, and at this time there are lowered temperature and electric irritability instead of the primary exaggerated condition. The bladder and rectum are subsequently affected, and various degrees of deranged function may be noticed. One of my patients is obliged to pass

<sup>1</sup> Op. cit.

<sup>2</sup> Manual of the Nervous Diseases of Man, Syd. Trans., vol. i. p. 267, *et seq.*

<sup>3</sup> Anatomie Pathologique, livre xxxviii. p. 9.

his water every ten or fifteen minutes, and his bowels are so constipated as to require an injection every day. The individual generally loses his desire for sexual gratification if the disease is at all advanced, though in the beginning there may be a marked disposition to erection. Muscular atrophy takes place if the anterior horns be affected.

An increase in the tendinous reflex is shown very markedly, especially if the gray matter of the cord be affected. The dorsal clonus is quite violent and the slightest tap upon any of the muscles causes a series of convulsive movements of great violence. The jarring of the patient will even give rise in some instances to an irregular coarse tremor of the lower extremities, which may last for several seconds. The invasion of the lateral columns is symptomatized by contractures, great spastic rigidity and discomfort. The legs and thighs may be so drawn up that the heels may make painful pressure upon the buttocks, and the contact of the knees when the adductors are the seat of contracture give rise to skin changes, and even ulcers. I have repeatedly found a "glazed" boggy skin readily pitting upon pressure, though the skin is usually of a muddy white color and either clammy or even dry and scurfy. Ferrier discovered a peculiarity in this disease due to skin changes; that if a *silver* coin was rubbed upon its edge a dark line would remain for some time.

**Causes.**—The common causes of myelitis are injury, syphilis, acute diseases, exposure, and extension of meningeal disease. Falls and blows upon the back are the origin of the majority of cases, but I consider syphilis to have a very great deal to do with even these, when often it is not suspected. Meningeal thickening or acute meningitis undoubtedly play an important part as mechanical factors; and in many cases reported, disease of the vertebræ has been found to produce the myelitis. Potts' disease seems to be a fruitful cause of myelitis and usually of a very serious variety. When so produced the atrophy and contractures of the limbs and active motor phenomena point to a decided implication of the antero-lateral columns of the cord. In such cases it is rare for the meninges to escape inflammatory action, and as a consequence, the symptoms of meningitis are added to those of the myelitis.

The existence of a large aneurism of the aorta, may also by erosion, prove to be a source of injury to the cord, and in some cases it is necessary to use great caution in making a diagnosis. In a case recently under observation, the gradual development of an irregular paraplegia was accidentally found to be associated with the presence of an abdominal aortic aneurism of large size, which produced a great deal of pain. There is a variety of myelitis which deserves the most careful study, because of its medico-legal importance, and I allude to that following spinal concussion. Cases of "railway spine" are so common in these days of railroad accidents, and there is so much danger of malingering, that I must add a word of advice to those who have occasion to go into courts of law as experts. That inflammation of the cord may follow a concussion, I think there can be no manner of doubt, and some of the cases of Erichsen support this theory; there are many others, however, in which hysteria plays



so important a part as to lead the examiner astray, unless he is prepared to avoid the error of accepting the patient's recital of subjective symptoms as conclusive. I do not think that any jury should give damage unless some physical signs of actual spinal disease are present.

The production of spinal inflammation from injury which does not produce external wounds, need not be of immediate appearance. It may be masked at first, but with due care it should be detected much earlier than Erichsen is disposed to grant. When present, the symptoms are conspicuous because of their irregularity and behaviour. Of the persons applying to the courts for redress, there are few who have suffered from early acute symptoms, but the cases are peculiar and therefore difficult to examine. In many of them unequal atrophy of the limbs, increased tendinous reflex activity, and ocular changes are present, while all are likely to complain of dysesthesia, loss of memory and mental feebleness, and incapacity for work. In those who sham, it will be found that there is an utter absence of physical changes, the tendinous reflex is neither exaggerated nor absent, the muscles respond well to electric stimulation, and the story of aches and pains is out of proportion with any possible kind of spinal trouble. The loss of memory and enfeeblement for brain work rarely stand the test of critical examination, and the patient's antecedent history does not bear out his story.

Venereal excesses, onanism, and continued dissipation are direct causes which should not be overlooked.

**Morbid Anatomy and Pathology.**—When the vertebral canal is opened, the investing membranes slit up, and the cord exposed, it will be found to be greatly changed in color and consistency at certain parts, and it may be diffuent and of a pinkish color. Scattered throughout the softened portion collections of blood may sometimes be found, and these are more often in the greatly altered gray substance, from which the disease seems to have started. At other points there may be discovered evidences of slight vascular changes, such as occur in the red stage of cerebral softening. There may be adhesions of the meninges to the cortex or collections of pus between them. In the more slow form of degeneration (chronic myelitis) the process may not be so widespread, limited areas being only affected. As the result of either form there may be an atrophic condition of the cord, or an actual hardness which we shall presently speak of in our consideration of sclerosis. The microscopical appearances are the following: the vessels are enlarged, varicose, or broken, and are surrounded by effused hæmatine; the nerve-tubes are swollen, irregular, and disrupted, and the axis cylinders substituted by oil-globules or granular debris; and the nerve-cells may have been broken down and become simple granular masses of a round or ovoid shape (Gluge's corpuscles). Fat globules may be found scattered here and there if the cord of an advanced case is examined; and the connective tissue may be found to be thickened and increased in density. Pus-corpuscles may also be seen. Dr. R. T. Edes, who, with Dr. S. G. Webber, of Boston, have done so much pathological work in the field of myelitis, presented a case to the American Neurological Association, which presented a not uncommon microscopical

appearance. The myelitis had lasted four months, and while the white matter was unaffected Edes found the gray nervous substance to contain little vacuoles in the anterior horns. The ganglia cell processes were shrunk and broken. Putnam, of Boston, had seen a case presenting the same appearance, and in his observations, there were collections of fat in the ganglion cells, and he was disposed to regard this deposit as indication of an earlier stage of the same process, which ended in Edes' case by the formation of vacuoles. In fact, he found openings at a lower level.

Jaccoud<sup>1</sup> speaks of two kinds of myelitis—*myélite en foyer* and *myélite central*. In the first form the meninges will be found to be injected and adherent to the nervous substance, and the nodules or patches may be several centimetres in length or smaller. These *foyers* are quite distinctly separated from each other by healthy tissue, and when one is removed the nidus in which it has formed is seen to be in quite normal condition. The anterior columns and anterior nerve-roots are often found to be involved; and the latter are the seat of “*pétites nodosités exuberantes*.” When the disease assumes a chronic form, these softened patches may become encysted as in cerebral softening. The *central* form, as its name implies, begins in the gray matter, and generally extends longitudinally.

<sup>2</sup>Dr. Gowers gives a most comprehensive diagram for the localization of spinal disease which I have reproduced (Fig. 37). It is founded upon anatomical and pathological data, and will enable the student to fix the level of the lesion by a consideration of the anatomical significance of the symptom.

**Diagnosis.**—It is necessary to exclude *spinal meningitis*, *locomotor ataxia*, *spinal tumors*, and *spinal congestion*.

*Spinal Meningitis.*—What I have already said in a previous article renders further consideration unnecessary.

*Locomotor Ataxia.*—There is no paralysis of motion in this disease, but rather an increased muscular activity, which is expressed by the violent manner in which the patient throws out his foot; while in chronic myelitis he drags one foot after another. The neuralgic pains in the extremities are absent in myelitis; while in locomotor ataxia they are marked symptoms. In myelitis there are none of the paralyzes of cranial nerves so commonly found with sclerosis of the posterior columns; the tendon-reflex is, moreover, usually absent in locomotor ataxia.

*Spinal Tumors.*—The presence of a spinal tumor may sometimes produce pressure upon the cord, and give rise to some of the symptoms. The slow development of the growth is, however, attended by corresponding slowly appearing symptoms, and the paralysis is not complete. The chance for doubt as to the condition arises when secondary myelitis results from such a tumor.

<sup>1</sup> Path. Interne, ed. 2me, vol. i. p. 310.

<sup>2</sup> The Diagnosis of Disease of the Spinal Cord, W. R. Gowers, M.D., F.R.C.P., London, 1880, p. 52.

Fig. 37.

|     | MOTOR. | SENSORY.                         | REFLEX.      |
|-----|--------|----------------------------------|--------------|
|     | 1 C    |                                  |              |
|     | 2      |                                  |              |
|     | 3      | Neck and Scalp                   |              |
|     | 4      |                                  |              |
|     | 5      |                                  |              |
|     | 6      | Neck and Shoulder                |              |
|     | 7      |                                  |              |
|     | 8      | Shoulder                         |              |
|     | 9      |                                  |              |
|     | 10     | Arm                              | Scapular     |
|     | 11     | Hand                             |              |
|     | 12     |                                  |              |
|     | 1 D    |                                  |              |
|     | 2      |                                  |              |
|     | 3      |                                  |              |
|     | 4      |                                  |              |
|     | 5      |                                  |              |
|     | 6      | Front of Thorax                  |              |
|     | 7      |                                  |              |
|     | 8      | Ensiform area                    | Epigastric   |
|     | 9      |                                  |              |
|     | 10     |                                  |              |
|     | 11     | Abdomen (Umbilicus 10th)         | Abdominal    |
|     | 12     |                                  |              |
|     | 1 L    |                                  |              |
|     | 2      | Buttock, upper part              |              |
|     | 3      | Groin and scrotum (front)        |              |
|     | 4      |                                  |              |
|     | 5      |                                  |              |
|     | 6      | Thigh { outer side               | Cremasteric  |
|     | 7      | front                            | Knee reflex  |
|     | 8      |                                  |              |
|     | 9      | Leg, inner side                  |              |
|     | 10     | Buttock, lower part              | Gluteal      |
|     | 11     | Back of Thigh                    |              |
|     | 12     | Leg and Foot { except inner part | Ankle clonus |
|     | 1 S    |                                  |              |
|     | 2      | Perineum and Anus                | Plantar      |
|     | 3      |                                  |              |
|     | 4      |                                  |              |
|     | 5      | Skin from coccyx to anus         |              |
| Co. |        |                                  |              |

FIG. 37.— DIAGRAM AND TABLE SHOWING THE APPROXIMATE RELATION TO THE SPINAL CORDS OF THE VARIOUS MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE SPINAL CORD. (From anatomical and pathological data.) (Gowers.)



*Spinal Congestion.*—These serious symptoms of myelitis are never produced by anything but a degenerative process, and there are rarely bed-sores, alkaline urine, or the profound disturbances of sensation or motion which characterize myelitis.

**Prognosis.**—In every case much depends upon the nature of the cause, and the extent of the cord involved. If there be a traumatism, of course this gives the disease a serious character, and death may occur in a few days.

Acute myelitis may run an exceedingly rapid course carrying off the patient in two or three weeks, and in such cases there are usually febrile symptoms. Webber<sup>1</sup> says, "It is not always easy to decide whether a case of myelitis should be called acute or chronic. The integrity of the whole cord is so essential to its proper function that if only a small portion is affected there are irregular and defective actions in all that part below, and perhaps in parts above. If an acute affection of one segment is recovered from with permanent injury of the diseased portion, the resulting symptoms are permanent, and there is chronic derangement of function. Inflammation may begin in an acute form in the lumbar enlargement, and then advance upwards slowly, yet pathologically, with the same characters in each segment of the cord; as no vital parts are affected, life is prolonged, and the cases seem to be chronic in time, while being acute pathologically. In fatal cases, then, the chronicity or acuteness depends upon whether vital centres are attacked early or late in the disease."

If the myelitis result from pressure from diseased and displaced vertebræ, the result, though more distant, is equally bad. Very few cases recover entirely from chronic myelitis, and in those that do, the lesion must either be due to syphilis, or be very limited.

The reparative action of a bed-sore is a valuable index of the central lesion. I have repeatedly witnessed the most varying and rapid changes, either on the result of an improvement, or the reverse in the diseased cord.

**Treatment.**—Counter-irritation, cold, and ergot are useful in the early stages of the acute disease. The former may be produced by the actual cautery, but care should be taken not to burn extensively, as the tissues are too ready to slough. Ice-bags may be used, and the patient should be laid on a water-bed, and kept as clean as possible; the thighs and nates being washed occasionally with salt and water, or with hot and cold water alternately. The iodide of potassium, with belladonna, should be given internally. Should the case be one of slow development, I prefer the use of ergot in half-drachm doses thrice daily; or we may use the bromides.

The sesqui-chloride of iron seems to have enjoyed deserved popularity in England, and it is preferred by Radcliffe to the iodide of potassium. In one case I obtained very excellent results with the tincture of the

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<sup>1</sup> Boston Medical and Surgical Journal, vol. cii., No. 7.

chloride of iron. Phosphorus and cod-liver oil, those valuable builders of nervous tissue, may be employed here with every hope that they will do good. In chronic myelitis they are especially serviceable, and small and frequent doses of strychnine are, in addition, useful. The use of the phosphate of silver has been so often followed by good results in recent cases, that I believe it should be tried, not only in this, but in other organic diseases. It seems to have a noticeably good influence upon the bladder, and in several cases I have found the patient was able to hold his water after its use.

There are forms of auxiliary treatment which not only increase the comfort of the patient, but go far towards ameliorating his disease. One of these is the assumption, if possible, of a position which shall favor the determination of the blood *from* the spine. Brown-Séquard has recommended that the patient should lie upon his side or belly, with his legs somewhat lower than the rest of the body. I have found that washing out the bladder with a dilute solution of carbolic or nitric acid, or chlorate of potash, prevents the disposition to cystitis which there very often is in myelitis. Warmth of the limbs, established by wrapping them in cotton batting, with a covering of oil-silk, or the new India-rubber tissue-paper, opposes contractions, and stimulates the cutaneous circulation; while application of the faradic current, and the employment of massage, help the patient to a great extent. The electric brush should be used faithfully every day, and it is better that the physician should make his own electrical application, than trust it to a nurse or attendant. The descending galvanic current of moderate strength may also be used daily.

### ACUTE ASCENDING PARALYSIS.

**Synonyms.**—Landry's Paralysis. Disseminated Neuritis (Gros). Progressive Paralysis (Graves). Paralysis ascendante aiguë (Dejerine).

**Definition and Symptoms.**—A form of advancing paralysis depending upon a rapidly developing central disease which affects successive portions of the cord in its upward course until it reaches the medulla, when death occurs. From the absence of any distinct anatomical change it cannot be said to be a myelitis. Westphall could not find any changes whatever in cases observed by him,<sup>1</sup> and Erb quotes various authors whose investigations have had the same result.

The disease begins by vague sensory changes referred to the extremities. There is an anæsthesia of the finger tips, so that the individual does not readily feel small things, and finds some difficulty in buttoning his clothes. He is indisposed to walk and grows easily tired, and this weakness in from one to six weeks increases to actual paresis, so that he becomes paraplegic and cannot walk at all. The disease seems to be confined almost exclusively to the motor tracts of the cord, and as the disease reaches a higher level we find a gradual loss of power in

<sup>1</sup> Abstract by Dr. J. J. Putnam in Boston Medical and Surgical Journal, Sept. 4, 1879, from original "Contribution a l'Histoire des Névrites, Paris, 1879."

the parts above. The muscles of the abdomen become weakened, and the functions of the bladder and bowels are much hindered, a resulting atony taking place. The patient, through weakness of the muscles of the trunk, is unable to hold himself upright (Erb), and as the intercostal muscles become affected we find various respiratory troubles, such as shallowness of breathing and dyspnoea. The arms in turn are paralyzed, and the muscles of the neck involved, and when the medulla becomes affected the symptoms of bulbar paralysis are presented, and the patient ultimately dies of asphyxia. Sensory troubles are very light, and occur only when the motor symptoms are well marked.

As negative symptoms may be mentioned—1. Absence of atrophy, except the slight amount resulting from inaction. 2. No abnormal increase of reflex excitability either cutaneous or tendinous. 3. No impaired susceptibility of the muscles to electric stimulation. 4. No contractions are ever present. Gros alludes to the varieties of the disease with reference to the duration and severity.

“There are three varieties: (1) the acute, usually fatal in the course of three weeks, often before the muscular atrophy commonly met with has had time (it is inferred) to develop itself; (2) the subacute, ending either in partial recovery or in death in the course of six months to a year, and liable, in the former event, to relapse; (3) the chronic, the most common form, lasting many years, but liable, also, to burst out into the acute variety at any time. The onset of the disease is commonly rapid, and not infrequently marked by a short febrile attack.”<sup>1</sup>

Gros considers the disease a centripetal affection, and calls attention to the tenderness at the peripheral ends of the nerves.

**Causes.**—The causation of the disease is not known, and all kinds of theories have been advanced—cold, intoxication, the poison of typhoid, diphtheria and small-pox have been alluded to as elements in its production, and syphilis has been suggested as a factor. The history of metallic poisoning would suggest the possibility that in some cases it might play an important part in the genesis of the disease. I know of one patient who died from acute ascending paralysis as a result of lead poisoning, and it is very probable that certain forms of acute paralysis following in the wake of the exanthematous fevers might reasonably be supposed to produce a peripheral neuritis.

**Pathology.**—The cord, brain and medulla have been repeatedly examined but without success, so far as the discovery of lesions were concerned. The sympathetic nervous system is probably primarily affected, judging from what Gros has said, and like some other form of spinal disease, in which primary changes appear in isolated groups of muscles, and which are supposed by modern investigators to be due to terminal lesions, so may this affection have a peripheral origin. Dr. Grainger Stewart<sup>1</sup> in an admirable paper upon a rare form of ascending neuritis, which, in many respects, resembles the disease under consideration, only

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<sup>1</sup> Edinburgh Medical Journal, April, 1881, p. 878.



in the trouble he describes there is an affection of sensory nerve fibres, as well as motor, and there are nerve changes. From the general character of the trouble he is inclined to believe the origin and pathology of the two diseases to be alike. This would point to the peripheral origin of acute ascending paralysis.

**Diagnosis.**—It is necessary to distinguish this disease from a myelitis which, if transverse, is symptomatized by decided affection of motion and sensation, and is attended by atrophy and decided disturbances of the pelvic organs, such as incontinence. Adult spinal paralysis is much more apt to be mistaken for the disease under consideration, than anything else, but here there is atrophy which is so decided and irregular as to be unlike the slight wasting of acute ascending paralysis. Gros speaks of the difficulty of distinguishing the disease from simple spinal meningitis, which even after all, may be connected with the affection under consideration. So far as my own experience goes there is enough muscular rigidity and spastic trouble to make a diagnosis, at least in the commencement.

**Prognosis.**—The duration of the disease may be very short; even three or four days may be sufficient for it to run its fatal course. Wilks<sup>1</sup> says, "In seeing such cases I am reminded of a spark alighting on a piece of touch paper, and the fire running through its length until the whole is quickly consumed."

Erb speaks more hopefully, and refers to Landry, who cured eight out of ten cases. In some cases the disease may come to a stand still for a time, and have a fresh outbreak, which carries off the patient. It is probable that the morbid process, whatever it is, may be of an exceedingly light grade, and affect the cord to a limited degree.

**Treatment.**—Active counter-irritation seems to have been most successful. This may be produced by the actual cautery or the application of croton oil. Cupping, faradization by the wire brush, and cold douches, certainly have done good in the German cases. Of course the use of remedies and food calculated to build up the nervous system, are to be employed, and among these are phosphorus and the fats. Cod-liver oil, the iodide of potassium, or the syrup of the iodide of iron, may be given alone or in combination.

## ANTERO-SPINAL PARALYSIS OF INFANTS.

**Synonyms.**—Paralyse essentielle de l'enfance (Rilliet and Barthez); Infantile Paralysis (Radcliffe Volkman, and others); Paralyse atrophique de l'enfance, Organic Infantile Paralysis (Hammond); Infantile Spinal Paralysis (Seguin); Spinale Kinderlähmung (Heine).

**Definition.**—This form of paralysis may be described as a condition usually characterized by a primary febrile stage, a secondary paralysis generally of the lower extremities, and a tertiary atrophy. The paralysis is incomplete, as sensibility is never lost.

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<sup>1</sup> Diseases of the Nervous System, p. 225.

**Symptoms.**—The disease is marked by a febrile onset of greater or less severity, attended by restlessness, malaise, and pains in the joints or back, and there may be rigors; or in some instances the loss of motor power is preceded by one or more paroxysms of convulsions. This febrile state is by many mothers mistaken for “teething,” “worms,” or other unimportant childish troubles, and it is not till the development of paralysis that any alarm is created. This symptom appears within two or three days from the beginning of the fever, and may take place at night. The only condition of disturbed sensibility is one of hyperæsthesia, which, however, is not a constant symptom.

Sinkler<sup>1</sup> has collected a number of cases in which he has noted the form of invasion of the disease. He found that the paralysis took place suddenly, that is, with prodromata in but 6 of 108 cases, while Dr. M. P. Jacobi<sup>2</sup> noted this form of invasion in 12 of 163 cases that she had collected. The modes of onset are the following:—

1. The child, while playing, suddenly drops palsied.
2. The child may be paralyzed at night.
3. Fever, but no convulsions; rapid loss of power.
4. Convulsions, followed by sudden paralysis. Sinkler reports but one case of this kind, and but two in which convulsions *followed* the paralysis).
5. The paralysis preceded by one for the exanthemata, or by whooping-cough.
6. Gradual development, perhaps limping at first, and afterwards complete paralysis, but no acute symptoms.

In this exceedingly valuable lecture, Sinkler throws much light upon the symptomatology of the disease, and gives the details of a classical case.

The paralysis may take the form of hemiplegia (Barlow and Duchenne have found cases of true cerebral hemiplegia, and Barlow has reported five such cases), or it may affect the voluntary muscles of all four extremities, and some of those of the trunk; but the facial muscles, as a rule, escape. After a short time there is a return of power in many of those at first involved, and but a small number of muscles (notably the anterior tibial, peroneal, and others of the leg and thigh) remain powerless.

The temperature of the paralyzed muscles is much lowered, and there is sometimes a difference of from eight to twelve degrees between the affected and normal sides. Heine considers the local reduction of temperature in old cases to be from ten to twelve and a-half degrees Fahrenheit. The bladder and bowels escape the paralysis, and their functions are consequently unimpaired.

Muscular contractility is lost with the commencement of the paralysis, and the faradic current will rarely produce contractions. Such, however,

<sup>1</sup> Clinical Lecture, Med. and Surg. Reporter, March 10, 1877.

<sup>2</sup> Am. Journ. of Obstetrics, May, 1874.

is not the case with the galvanic, except in extreme instances, or when the case is one of long standing. So far there are rarely any evidences of atrophy or contracture of the paralyzed muscles, but it will be found now that certain muscles at first affected begin to regain their lost functions, while others become atrophied and utterly useless. Even the galvanic current fails to stimulate them; and at this period, which may vary from four to five weeks to six months from the beginning of the disease, there may be deformities and muscular contractures, which may result either from the weight of the body upon the affected limb, or from the antagonism of non-paralyzed muscles; but Volkmann<sup>1</sup> considers that this incapacity of the limb to support the superimposed load is of much greater importance as a cause of deformity than the mere antagonism of the unaffected muscles.

The foot is apt to drop so that the toes hang limp and flaccid. Barlow alludes to the "*talus pied creux*," a deformity described by the French writer, the instep being prominent and the sole hollowed.

Such deformities may take place as lateral curvatures of the spine, talipes, and other distortions which appear as various muscles are paralyzed, or, if there be shortening of the limb (which is by no means uncommon), as a consequence of reduction in the length and size of bones which have become atrophied. The deformities that may result from the disease under consideration are of a *primary*, and of a secondary or *compensatory* nature. The primary forms are those which are seen as talipes of both kinds, and result from loss of sustaining power of the muscles. The *compensatory* consist in spinal curvatures, such as lordosis or scoliosis.<sup>2</sup> The skin is usually blue and livid, and the temperature is much below that of the healthy limb. These deformities rarely disappear, but continue throughout life, which is in no way shortened by the disease. The following cases may be presented to illustrate the appearance and behavior of the disease. The first case is somewhat anomalous, as there were two forms of paralysis; the primary attack being hemiplegia, and the secondary paraplegia.

CASE I.—Robert B. (a seventh-month child) was sent to me by Dr. H. G. Piffard, of this city. During September, 1876, he became feverish, and, after two days, during which he was confined to bed, he had a general convulsion. Before his fever he had eaten a great quantity of cherries, and his mother supposed his illness to be due to this cause. The mother stated that the convulsion lasted three and a half hours. He became paralyzed two days afterwards, the right arm and leg being affected; but two days after this he could use even these limbs. A few days subsequently he went out to play, but came back feeling out of sorts; and, after a few hours' fever, another spasm took place. Within the next thirty-six hours both legs were paralyzed, so that he could not stand. Towards the first of November he regained some power, and can now stand when holding a chair.

<sup>1</sup> Sammlung Klinischer, Vorträge, Heft 1, 1870.

<sup>2</sup> Produced by attempts to restore disturbed equilibrium.



*Present Condition.*—He is a puny boy, about five years old, and is badly nourished. He has no voluntary power over lower extremities, but can move the arms perfectly. The legs are both very much reduced in size, and the muscles are flabby and atrophied. The peronei, solei, and anterior tibial muscles are reduced in size, and have lost their electric contractility. He perceives pinches, and changes of temperature, and the “wire-brush” produces much pain. The skin is cold, mottled, and dry, and here and there is dotted with patches of scurfy eruption.

CASE II.—Annetta F., aged 10 years. About three years ago she became quite ill after a sleigh ride, and it was supposed that she had “caught cold.” Her feverish symptoms were quite decided, and she was slightly delirious. After several days she seemed to improve slightly, but on awaking one morning it was found that she was paralyzed and unable to rise; and she complained of intense backache and tingling of the limbs, which, however, were of very short duration. About two months after this she began to recover the use of her arms, but the legs were more fully paralyzed; and it was several months before she began to move her toes, and finally made feeble movements of a more extended character. The muscular contractions of the flexors were performed more easily than movements requiring extension; and, after a time, she attempted to walk, but at first this act was impossible. During the next year she was obliged to use crutches, and needed the assistance of her nurse. When I saw her, there was talipes equinus varus of the left foot, while the right seemed to be but little affected. Flexion was possible, but extension of the leg or foot was beyond her power. There was some relaxation of the ligaments of the knee-joint, so that when I made extension I caused the tibia to form an obtuse angle with the femur, so that there was some anterior curvature. Her gait was peculiar, and she swung the left leg, bringing it down with a jerk. The skin covering the left leg was dusky and mottled, and seemed in close contact with the tissue beneath; and the surface-temperature was several degrees below that of the other side. No rectal trouble,

CASE III.—A girl sent to me by Dr. Lockwood, of Norwalk, had presented, among other symptoms, mitral disorder, fever, general paralysis, residual paralysis, paraplegia, and paralysis and atrophy of the right deltoid, which cannot be made to contract when subjected to either current. Right leg more affected than the left.

CASE IV.—A girl 10 years of age. At the second year after a fall she became feverish, was delirious, and took to her bed. There was general paralysis of the right leg and thigh; but after three months there was improvement, except of the leg, which remained paralyzed. There are now a pronounced talipes varus, complete atrophy of the anterior muscles, and utter loss of electro-muscular contractility. She has used various forms of orthopædic apparatus without relief.

CASE V.—Frank N. C., 4 years old, a stout, rugged boy, enjoyed good health until January, 1877, when he contracted scarlet fever, with albuminuria as a result. From this he recovered, but in August he again fell sick with what was pronounced to be rheumatic fever. There were high temperature, some diarrhoea, which lasted for a number of days, painful joints, and loss of power in both lower extremities. The power returned in the right leg, so that by the middle of September (three weeks from the invasion of the fever) he had control of that member. The left remains powerless, and there has been slow atrophy. The exten-

sors of the leg and foot are now powerless, and there is decided atrophy of these and the posterior tibial, abductors of the thigh and anterior muscles. The knee-joints are quite weak, and there are projections on the inner side of both knees. He is knock-kneed, no eversion or inversion of feet, but there is slight talipes of the left foot.

CASE VI.—Mamie W., 6 years and 1 month old, always was a nervous, excitable child. Has had several convulsions in her life of an epileptic character, without any after-effects, or apparent coexisting disease. In July last she had whooping-cough. On September 4th she was taken with colic, malaise, and convulsions, during which the body became rigid, and she frothed at the mouth. These convulsions appeared at 5 P. M., and lasted until midnight. She was unconscious all the time. At 7 P. M. the corner of the mouth became drawn up by spasms. She had fever during the following day and for a number of days. Did not make any attempt to move for a number of days, and for twelve days she could not speak. She was found to be *generally* paralyzed, and after a short time the arms recovered their strength, but the legs began to lose their size and shape, and became smaller than they were before. Her mental condition is defective (five weeks after attack). And, though there is no impairment of bladder or rectum, she does not call attention to her wants, but defecates and urinates in her clothing. Power of upper extremities good. The legs are cold and mottled; there is slight talipes on both sides; and great wasting of the flexors of the feet, especially of the right. Faint contractions are excited by the strongest faradic currents, but she can move her toes very feebly, but not flex the foot. She has control over the thighs. Both feet are slightly everted. There is redness of the skin covering the right knee, but no pain; no pain in back; slight impairment of sensation, but reflex irritability not embarrassed, as was demonstrated by pinching; pupils moderately dilated.

The muscles of the leg are more often affected than those of any other part. In nearly every instance the *tibialis anticus* is paralyzed, and in 18 of the 23 examples I have noticed this muscle was affected. The *peroneus tertius*, *longus*; *extensores longi digitorum*, *proprius pollicis*; and the *flexores longi digitorum*, and *longus pollicis*, are usually affected. The deltoid is paralyzed more rarely, and of the cases I have enumerated there were but two in which this muscle was affected. The muscles of the upper extremities are seldom involved in comparison with those of the leg, and those that are usually paralyzed are the flexors of the hand. Though the muscles of the trunk may be sometimes involved in the early paralysis, it is extremely rare that we find any residual paralysis of any of them. Barlow and others have witnessed repeated attacks of paralysis in the same subject after apparent complete recovery.

It is rare to find either arthritic enlargement or wasting, or bed-sores in uncomplicated essential spinal paralysis; but this disease, which is limited to the anterior columns, should not be confounded with a transverse myelitis or compression myelitis that may be found among children which are not always clearly distinguished, and give rise to tissue changes.

**Causes.**—The etiology of the affection is anything but clear. Exposure and bad or insufficient food are supposed to account for it, just as

they do for many other diseases of the same class. Barlow alludes to the fact that an unrecognized form of exposure arises from taking a child into a sleeping-room with newly-plastered walls. It is a significant fact that more of these patients belong to the lower walks of life than to the higher, and that the children of the destitute poor, who come of drunken parents, and are "knocked about" and half-fed, are those who are generally the victims of the disease. As to age, Sinkler has found that 84 of 108 cases were between the ages of six months and three years, and that half of this number were males. Barlow,<sup>1</sup> speaking of the infantile form, states that he found that there was no great preponderance of the disease in either sex, and that of 63 cases he had collected, 33 were males and 30 females. His other statistics show that the disease more commonly begins before the second year, and that 42 of the 63 cases occurred between the first and second year of life. It will thus be seen that Barlow supports the other authors I have mentioned. Of 53 cases in which the attack could be fixed with accuracy, 27 occurred in the months of July and August.

Duchenne<sup>2</sup> holds that two-thirds of the cases begin before the second year, which view I am disposed to take. Warm weather seems to favor the development of the disease, and in nearly two-thirds of Sinkler's cases the disease began in the months between May and October. Cases have been reported in which the exanthemata have preceded the paralysis, and varicella, measles, and scarlatina may be mentioned among these; but it is probable that in the majority of such cases sclerosis not limited to the anterior columns has been the central condition.

**Morbid Anatomy and Pathology.**—We are indebted to Charcot<sup>3</sup> and Joffroy, Duchenne,<sup>4</sup> Echeverria,<sup>5</sup> and others for reports of autopsies and microscopical examinations, and as the result of their investigations the following appearances may be looked for.

In the early stages of the disease there is probably a condition of sub-acute myelitis, with softening and destruction of nerve-elements, etc. This is confined exclusively to the anterior horns. Some of the nerve-cells of this portion of the cord are sometimes filled with granular pigment deposits, while others are disorganized and broken up. The nerve-tubes of the anterior roots will be found shrunken, the myeline absent, but the axis cylinder is nearly always intact.

In other cases of longer standing there are evidences of atrophy of the anterior horns, perhaps amyloid degeneration, and sometimes sclerosis. The nerve-cells are found in an atrophic condition, or absent altogether. The white matter of the anterior and lateral columns is not rarely the seat of such degeneration, and proliferation of the connective tissue is some-

<sup>1</sup> On Regressive Paralysis. W. H. Barlow, M. D., Manchester, 1878, p. 4.

<sup>2</sup> De l'Electrisation localisée, 3d ed., Paris, 1872, p. 417.

<sup>3</sup> Archiv. de Phys., tome iii., 1870.

<sup>4</sup> Ibid., tome iv., 1870.

<sup>5</sup> Reflex Paralysis, etc., p. 29, New York, 1866.



times found. In 25 cases, collected by Seguin,<sup>1</sup> the constancy of the lesion is very clearly shown.

|  |           |
|--|-----------|
| The anterior horns together were affected in . . . . .               | 11 cases. |
| The right anterior horn alone was affected in . . . . .              | 1 case.   |
| The left " " " " " " . . . . .                                       | 4 cases.  |
| Both affected in . . . . .   | 6 "       |
| Sclerosis of antero-lateral columns (chiefly) and other white matter | 13 "      |
| Tubercles and blood-clots . . . . .                                  | 2 "       |
| Meningitis and meningeal congestion . . . . .                        | 2 "       |

Damaschino<sup>2</sup> and Roger, Cornil,<sup>3</sup> Clarke,<sup>4</sup> Charcot,<sup>5</sup> and Joffroy have added many histories to those given to the profession by the early writers, and it is now well settled that the anterior horns and lateral columns are the seats of the central lesion.

Rosenthal<sup>6</sup> considers that the primary cause is dilatation and thickening of the vessels, and does not believe that the morbid process begins by degeneration of the nerve-cells. Notwithstanding the appearance of well-defined lesions in nearly every case, there are occasional examples of the disease where no central changes are to be found. Kétli<sup>7</sup> reports one of these in which extensive muscular alterations were visible, but not the slightest indication of central disease. Elischer<sup>8</sup> examined the muscles, which were seen to be the seat of both fatty and colloid degeneration. The sarcolemma and nerves were not altered. In the striated muscles, instead of the single normal cell-nucleus, there were seen three or four granular cell-nuclei, which seemed to be at the same time enlarged, and contained two or three, or even more nucleoli. The contractile material was diminished, so that it did not fill out the sheath, but drew away from it. This atrophy was so great that at the upper and under part of the spindle-shaped cell-nucleus of the sheath there was hardly to be found a breadth of .002 millimetre of cross-striped contractile muscular substance. Kétli thinks that these changes in the muscle without central disease point to the peripheral nature of the affection, in which opinion he has but few followers. Lesions of peripheral nerves have been found by various observers. Rinecker<sup>9</sup> reports an autopsy, made by Förster, in which these nerves were found to be thin, shrunken, and greatly degenerated. The bones and muscles present appearances which are perhaps more interesting than those of the cord.

The muscular fibres are at first found to be reduced in size, and subsequently the transverse striæ gradually disappear, while the longitudinal fibres become more marked. There is increase in the connective tissue, and next a fatty degeneration, the oil-globules taking the place

<sup>1</sup> Spinal Paralysis, etc., pp. 12-13.

<sup>2</sup> Gaz. Méd. de Paris, 1871.

<sup>3</sup> Med. Chir. Trans., vol. ii., 1869, p. 249.

<sup>4</sup> Op. cit.

<sup>5</sup> Ibid.

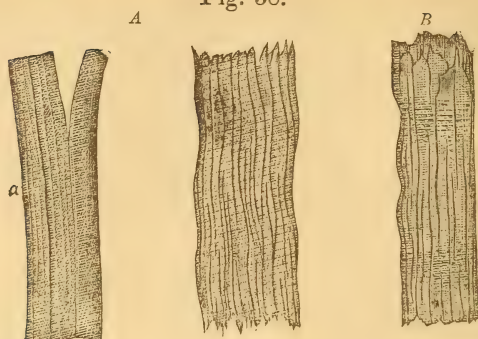
<sup>6</sup> Jahrs. für Kinderheilkunde, 1871, 5 Heft 1.

<sup>7</sup> Ibid., 1864, p. 290.

<sup>8</sup> Quoted by Fox, op. cit., p. 290.

<sup>9</sup> Ibid.

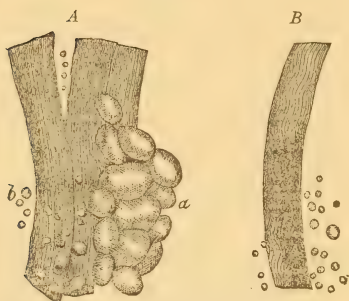
Fig. 38.



*a.* Normal fibre.

*A.* Represents the normal fibres with well-marked transverse striæ. *B.* The transverse striæ are not quite so distinct, but the longitudinal fibres are well marked.

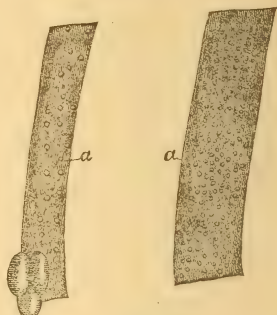
Fig. 39.



*a.* Fat cells. *b.* Interstitial fatty deposits.

The stage of fatty degeneration. *A.* The longitudinal fibres are only seen, and there is a deposit of round and oval adipose cells and oil-globules. *B.* Undulations of longitudinal fibres.

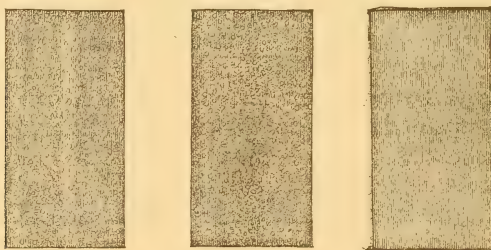
Fig. 40.



*a. a.* Fat molecules.

The progressive fatty degeneration and the disappearance of longitudinal fibres.

Fig. 41.



This illustration represents the final stages, in which it will be seen that the muscular fibre has lost its identity, and at last there is an absence even of oil-globules.

of the normal muscular tissue, and finally nothing remains but the connective tissue and fat, which latter disappears, leaving the sarcolemma bound together by connective tissue.

The accompanying cuts, from Duchenne, show the changes that take place.

The blood vessels running to the atrophied muscles are often of smaller size than they should be, and sometimes are the subject of atheromatous degeneration.

The bones also undergo atrophic changes, becoming friable and thin, and occasionally the seat of fatty degeneration. The cartilage covering their articular extremities is roughened, and in some places detached.

Though some observers have maintained the peripheral origin of the disease, the large majority have adopted Heine's original views advanced in 1840, and endorsed by Duchenne in 1855. The almost general opinion that the disease is of central origin has been conclusively proved, I think, by the large number of autopsies, the most valuable of which have been made in late years.

Westphal's views in regard to the existence of trophic cells, which were also adopted by Duchenne, certainly receive decided confirmation in the constant atrophic processes which are connected with degeneration of the cells of the anterior horns.

That it is not a disorder dependent upon the sympathetic system has been proved by the utter absence of any diseased condition either of the ganglia or the nerves.

**Diagnosis.**—The existence of febrile symptoms, and the secondary complete paresis which changes its character and is finally confined to a few muscles, the unimpaired sensibility, and the rapid sequence of atrophy and deformities give this disease a distinct character which does not admit of any mistake in diagnosis. Forms of reflex irritation, such as ascarides, adherent prepuce, and like peripheral conditions may produce some of the symptoms, but their non-progressive character, and disappearance with the removal of the cause, should make the possibility of an error very remote.

**Prognosis.**—Much depends upon the behavior of the muscles under electrical stimulus. If the least response either to the galvanic or faradic currents can be recognized, the chances are extremely good, and it only remains for the physician to be patient and attentive. In regard to duration and its bearing upon prognosis, I may state that many cases have been cured even after deformities have taken place. Klopsch,<sup>1</sup> of Breslau, reports several of these cases. In one there was shortening of the thigh and deformity of the pelvis, as well as other serious troubles. Much of the hope of cure, however, depends upon the care taken in the treatment.

**Treatment.**—The most active and useful agent in the therapeutics of this disease is undoubtedly electricity, either as galvanism or faradism, applied to the muscles. The treatment of the central lesion is also of importance, and it is advisable to begin an energetic course of ergot, with the actual cautery, before the atrophic condition commences.

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<sup>1</sup> Ullsburger's Prize Essay, *Am. Journ. of Obstet.*, 1870-71.



After this the central disease is very difficult to manage. Heine recommended strychnine, which, in young children, may be given in doses of  $\frac{1}{120}$ th of a grain, and afterwards increased. Cod-liver oil and sea-air, good food, and tonics are of as much importance as anything else.

When we come to the treatment of the paralyzed muscles, we may try electricity, massage, hypodermic injections of strychnine, and the application of heat and cold. If the faradic current be found to be incapable of producing contractions of the paralyzed muscles, we must make use of the galvanic. From ten to thirty<sup>1</sup> cells of any good galvanic battery should be employed, and the electrodes must be covered with sponge or cloth. When the positive electrode is placed in the groin (if the legs are paralyzed), and the negative over the muscle or muscles paralyzed, a contraction may be seen; if such does not take place, the current may be slowly intermitted by proper apparatus, or by simply removing the sponge from the surface and reapplying it again. If the current be too strong, or if the application be too protracted, we may be disappointed, for the small amount of electric irritability that exists may be quenched before an appreciable contraction is perceived. It is therefore better to use a current of low tension. If we are gratified by the appearance of a contraction, we should produce two or three more and then stop for the day. By increasing the muscular stimulation little by little each day, we may finally create powerful contractions with a minimum current, and after a short time we may substitute the faradic current. It is of great importance that muscular relaxation should be produced during the use of electricity. I may repeat what I have already said, and add that a tired muscle naturally responds less perfectly to electric stimulation than one which is unimpaired. If massage is used, it is well to knead and rub each muscle every day.

Should electricity fail to relieve the contracted condition of the limbs, which may be present, we may avail ourselves of the knife. Tenotomy is often of service, but it should not be prematurely resorted to, but left as a last resource when all other remedies fail. Various methods for improving the temperature of the paralyzed limbs have been described by Roth.<sup>2</sup>

In brief they are the following:—1st. The position should be attended to in all cases; a paralyzed part should not be permitted to hang down, and to dangle about; it should be placed in a horizontal position, and the coldest part should be the highest, which assists the reflex of venous blood.

2. Clothing.—Spun silk, a mixture of silk and wool, wool or fur garments should be worn next to the skin; it is only in exceptional cases that the hyperæsthesia of the cutaneous nerves does not permit any of these materials to be used. Here silk is placed next to the skin, and wool

<sup>1</sup> It will rarely be found necessary to use this number, and it is advisable to begin with the weakest current that will provoke contractions.

<sup>2</sup> On Paralysis in Infancy, Childhood, and Youth. London, 1869, p. 83, ch. 62, quoted by Barlow.

or fur over it. The paralyzed part should be well warmed before it is covered with bad conductors of heat. Roth recommends also exposure of the leg to direct heat of the fire, a screen with a hole for protection of the rest of the body to be provided. He also recommends the use of Turkish baths, the application of a bag filled with hot salt or sand, and the usual form of massage and electricity to which I have before alluded.

Volkman speaks in glowing terms of the use of Junot's boot, which, with the rubber muscle of Sayre, and the plaster bandage, is a useful form of treatment in cases of long standing. The paralyzed limb is placed in the boot and the air exhausted, so that a determination of blood to the part shall be induced.

## ANTERO-SPINAL PARALYSIS OF ADULTS.

**Synonyms.**—Acute anterior spinal paralysis. Subacute general anterior spinal paralysis (Duchenne). Spinal paralysis of adults (Meyer, Charcot, Gombault). Myelitis of the anterior horns (Dujardin-Beaumetz, Seguin). Acute spinal paralysis of adults (Petitfils). Anterior poliomyelitis (Erb, Eisenlohr). Acute anterior poliomyelitis (Kussmaul).

Fig. 42.



Antero-spinal Paralysis.  
(Seguin).

**Definition.**—A myelitis of the anterior horns of the spinal cord, either symptomatized by an acute invasion attended by fever, and followed by sudden paralysis, or by the gradual appearance of the paralysis which becomes complete and next partially disappears, leaving certain muscles affected; unattended by loss of sensation, or vesical and rectal trouble.

**Symptoms.**—I am indebted to the little memoir of Dr. E. C. Seguin for assistance in the preparation of this article, and for the report of a case which afterwards fell under my observation when I followed him as visiting physician to the Epileptic and Paralytic Hospital. Duchenne<sup>1</sup> first called attention to this form of paralysis as early as 1853, and recognized its identity with infantile paralysis. In 1863 Charcot<sup>2</sup> was struck with the similitude between the two diseases, and in 1872-73 and later years Gombault,<sup>3</sup> Dujardin-Beaumetz,<sup>4</sup> Petitfils,<sup>5</sup> and Bernhardt<sup>6</sup> have presented cases, and decided the fact that infantile

<sup>1</sup> De l'Electrisation localisée, Paris, 1872, p. 437 et seq.

<sup>2</sup> Papers of Petitfils.

<sup>3</sup> Archiv. de Physiol. norm. et path., 1873, pp. 80-87.

<sup>4</sup> De la myélite aiguë, Paris, 1872.

<sup>5</sup> Considération sur l'atrophie aiguë des cellules motrices, Paris, 1873.

<sup>6</sup> Arch. für Psych. und Nervenkrank, 1874.

paralysis had an analogue in adult life. Gombault brought forward the first case with an autopsy confirming the theory enunciated by Duchenne, and in this country the admirable little works of Seguin epitomize all that has already been brought forward. The first case seen by Seguin<sup>1</sup> has since fallen under my observation, and from his published notes I copy her history.

Female, unmarried, aged twenty years. Admitted to the Epileptic and Paralytic Hospital, Blackwell's Island, service of Dr. E. C. Seguin, November, 1871. Patient presents a paralyzed and extremely atrophied left leg, and gives the following imperfect history: The trouble began nine months ago, suddenly during sleep, with painful contractions; she then gradually (?) lost power in the left leg; no other limb affected. The patient cannot state how long a time elapsed between the first symptom and the discovery of palsy. She adds that, on the day before the attack, her left leg felt quite cold and a little numb; and that her menses were suppressed. No cause is apparent—no hereditary influence, no injury.

Examination: Left foot is drawn up in moderate *pes equinus*, with inward inclination. No voluntary movements below the knee. The patient's answers to the æsthesiometer test are unreliable; sensibility to painful impressions is somewhat impaired, that to temperature preserved; tickling is felt equally on both feet. Pressure shows tenderness over the lumbar vertebræ; no spontaneous pain. The right calf measures 26.9 c. in circumference, the left 23.7 c. There is absolute loss of electro-muscular contractility in all the muscles of left leg. The left leg is very cold, and its circulation feeble. I frequently called the attention of the resident staff and of friends to this remarkable case as one of the same kind as that which, occurring in the early years of life, we call infantile spinal palsy.

The subsequent history need not be reported. No treatment did any good; the girl remained in the hospital without any acute symptoms, and went away October 3, 1873, carrying this wasted left leg. She was employed as a help in the wards of the Convalescent Hospital on Hart's Island, and was there much exposed to cold.

The second attack, of which patient gives a good account, came on late in December, 1873. Had pains "like rheumatism" in right leg; there was a feeling of pins and needles in the limb, this numbness extending above the knee. She is positive that on the fourth day the right leg was completely paralyzed. No symptoms in left leg. No bed sore, and no affection of bladder or rectum. Re-admitted to the Epileptic and Paralytic Hospital, March 3, 1874, with atrophy and palsy of both legs; no acute symptoms.

During the spring and summer this patient rather gradually lost strength in the thighs, in the right most. She also exhibited a variety of interesting visceral disturbances, consisting of amenorrhœa, lasting two or three months; the menses then appearing with much pain, the blood abundant and in clots; there were also pains in the back and lower abdomen. On many days in this period the urine had to be drawn off with the catheter, and it often was bloody, exhibiting a heavy mucous deposit, and containing albumen. The microscope showed only leucocytes and a variety of epithelial cells—there being probably both pyelitis and cystitis.

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<sup>1</sup> Spinal Paralysis, N. York, 1874, and Anterior Myelitis, 1877.



Since the middle of September has not required the catheter, and, with exception of palsy, has been better.

Re-examined October 25, 1874. Patient, when she first came in this year, walked ill with a crutch and stick; is now able to walk with two sticks (result of education). Cannot stand or walk without help. The patient is a stout and healthy girl, exhibiting nothing abnormal above the hips. Both lower extremities are extensively palsied and much wasted. The left leg (first attacked in 1871) shows no voluntary movement below the knee, with exception of slight separation of the toes. As the patient lies on the bed she is able to raise the extended limb as a whole; but the strength at knee-joint is small. The thigh is thin and flabby; the leg is the seat of extreme atrophy, and looks just like the same part in cases of infantile spinal palsy, there being apparently only connective tissue and fat around the bones, the skin being bluish and very cold to the touch. The right lower extremity (paralyzed in 1873) is in a very similar though less extreme state. All voluntary movements are possible with the foot, though they are feebly performed. The limb, as a whole, cannot be raised from the bed, and flexion at knee-joint is weak. The quadriceps extensor femoris is wholly paralyzed; the flexors of the thigh upon the body act feebly; the adductors fairly. Both feet lie extended and adducted; toes flexed. The right leg is, like the left, extremely wasted, bluish and quite cold. Sensibility to contact, pain, and temperature are preserved in both limbs. Tickling is felt, but produces no reflex movement in the palsied parts. The electro-muscular reaction of the atrophied muscles of both limbs is lost (both currents). At present, urine is passed normally. The patient's arms, shoulders, and chest are large and rounded, standing in remarkable contrast to the dwindled legs. There have been no bedsores and no spinal epilepsy.

|  |         |
|--|---------|
| Circumference of right thigh (lower third) | 31.5 c. |
| “ left “ “                                 | 30.5    |
| “ right calf                               | 24.0    |
| “ left “                                   | 21.5    |
| “ forearms                                 | 25.0    |

On a healthy girl (non-palsied) of same proportions as the patient, the following measurements are obtained:—

|                             |         |
|-----------------------------|---------|
| Circumference of right calf | 35.0 c. |
| “ left “                    | 34.5    |
| “ forearms                  | 24.0    |

The patient having been in bed some time, well covered up, has a thermometer held between the great and second toes of each foot for three minutes, with results:—Right side, 84.25° Fahr.; left side, 86° Fahr.

In March, 1876, the patient came under my charge, when I found that her condition was somewhat aggravated. She manages to go about with the aid of crutches, but has *utter* loss of power below the knees. The tactile sensibility is much lowered, and tickling can be borne without any reflex movement being produced, and she has lost control to a great extent over the bladder and rectum.

Another case reported by Lincoln is well worth presenting, as illustrative of this form of disease beginning without fever.

A tall, stout man,<sup>1</sup> 49 years of age and of previous good health, noticed one morning, without any previous symptoms, a feeling in his legs as if they had fallen asleep. The feeling came on again and again through the day, and he began to be a little weak in the legs. In the afternoon, when trying to step upon the platform of a street car, he failed, and had to be helped in. On arriving home, he was able (with assistance) to walk up stairs to his bedroom, and went to bed, where he remained.

When seen by Dr. L., two days later, he felt well, no giddiness, muscles of face and eyeballs under perfect control, pupils normal in size and contracted well, speech natural, vision and hearing without defect. The bladder and rectum performed their functions normally. The senses of touch, pain, and temperature were normal in the hands, and nearly so in the feet. Reflex contractions could scarcely be obtained from the soles. There were no abnormal sensations. Pulse, 80; temperature, 98°. No albumen in the urine.

The muscles of the neck and limbs, except below the knees, were generally in a condition of semi-paralysis. He lay on his back almost helpless; could not raise his head from the pillow without some help, and could not raise his knees from the bed by flexing the thighs. The grasp of his hand was very feeble indeed. There was no paralysis of any muscle. Below the knees he seemed to have more strength. The weakness was much more marked on the left than on the right.

Treatment consisted at first in *nux vomica* and *cinchona*, and subsequently tincture of iron with *strychnia*, and *Horsford's acid phosphates* of lime and *magnesia*. On the fifth day of the attack, treatment by the induced electric current was begun, when it was found that some at least of the muscles had lost part of their susceptibility to this stimulus. The loss went on increasing until the twenty-first day, when the galvanic current was substituted, a descending current being applied to the spine, and interrupted currents to the muscles, three times a week; the faradic current was also continued for a few weeks.

The hot-air bath to profuse perspiration was used just before the application of the currents, together with regulated gymnastic exercises. The paralysis of the muscles was gradually relieved under this treatment to a very considerable degree. The patient's improvement was very gradual, and it was six months before he was able to ride out. He finally was enabled to attend to his business pretty much as before the attack.

Other cases begin much more slowly, and several of this kind are reported by Duchenne, but the origin of the disease is nearly always sudden. There may be pain or dysæsthetic symptoms, or no warning at all, the patient awaking in the morning and finding himself paralyzed, as was the case with Seguin's patient. Like the infantile form, there may be an acute attack of fever, which may last for several days, during which there is usually delirium or rigors. The paralysis appears during this time, and may be general, so that the upper and lower limbs are affected and the loss of power is complete. The functions of the bladder

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<sup>1</sup> Boston Medical and Surgical Journal, March 25, 1876.

and sphincter ani are always normally performed until other parts of the cord are affected, and there is neither incontinence of urine nor involuntary evacuations. At the end of a few weeks there is a commencing improvement, some of the muscles regaining their lost power and contracting quickly under electric stimulus, while atrophy of those already paralyzed begins to take place. The skin over the paralyzed limb is quite cold and blue, and there is diminution of temperature and faradic excitability, while ultimately it is impossible to provoke any response, and the limbs become deformed and twisted. Atrophy of deeper parts follow, and the bones become reduced in size, while the articular ends appear large in contrast with the attenuated size of their shafts. Sensibility is rarely disordered, though exceptional cases of anæsthesia or hyperæsthesia are met with, but after the inflammation has involved the posterior columns the phenomena of general myelitis are presented. Dysæsthesiæ are common, and the patients complain of subjective cold, various pains, and the waist-constricting band. The muscles of the face, neck, chest, and abdomen are rarely affected, but the extremities remain deprived of pain after there has been a considerable retrocession of the original complete paralysis. The atrophy is rapid, and differs from that of progressive muscular atrophy in the fact that whole groups are affected at a time, while the peculiarity of progressive muscular atrophy is that muscles are irregularly affected. There are never bedsores.

The disease may be so rapid in its development as to suggest the malady known as acute ascending paralysis, and it is probable in such case that the extension of the disease proper is not always confined alone to the anterior columns.

Erb<sup>1</sup> alludes to a light variety of spinal paralysis, which has been described by Kennedy, Fry, and others. To this variety has been given the name "temporary spinal paralysis." The paralysis is characterized by its brief duration, and may involve a limited group of muscles or several groups. It would seem, therefore, that there are two varieties: the temporary and permanent; but Seguin and others have made the classification *acute*, *subacute*, and *chronic*, which is based rather upon the variety of myelitis than the paralysis. Duchenne applies the term *sub-acute* to the former, which begins without fever, attacks the lower extremities first, and, extending upwards, involves the muscles of respiration and deglutition.

**Causes.**—The same unsatisfactory history of exposure, fatigue, and peripheral irritation is connected with the history of this as well as other spinal diseases. In four of Seguin's cases surface exposure to cold is said to have produced the attack, and in three other cases, "refrigeration" is named, while in others dysentery, measles, and other acute diseases were at the origin of the trouble.

As regards age and sex, I can do no better than refer to the tables of Seguin. All of the patients whose histories he collected were of middle

<sup>1</sup> Archiv. für Psychiatrie, Band v., Heft 3.



age. "The greatest age at the time of seizure was 62 years, the least 18 years." Among 17 cases reported by various observers, there were 13 men and 4 women.

**Morbid Anatomy and Pathology.**—But very little light has been thrown upon the morbid anatomy of the cord, which accounts for this form of paralysis. Chalret<sup>1</sup> and Gombault<sup>2</sup> have reported two cases.

The appearances found may be briefly enumerated as these: The horizontal fibres which pass from the anterior horns to form the anterior spinal nerve-roots were diminished in size, and the large ganglion-cells of the anterior roots were atrophied, having undergone yellow pigmentation. Some of the nerve-cells which had not undergone this form of degeneration, were also reduced in size. This information is very meagre, though these two cases illustrate the pathological anatomy of the disease. Charcot and the majority of observers believe that the situation of the lesion is always in the anterior horns. The only matter of dispute seems to be whether or not there is primary degeneration of the cells, or an acute interstitial myelitis and secondary injury of the nerve-cells. This latter view is held by Erb,<sup>3</sup> and, I think, is being generally adopted.

The muscles were found to be in a state of fatty granulation, which is the case in the infantile variety. In some respects the disease resembles progressive muscular atrophy and bulbar paralysis, the lesion being atrophy of the motor and trophic cells, but it is probable that the trophic cells are primarily affected in these latter diseases.

**Diagnosis.**—Antero-spinal paralysis is likely to be sometimes mistaken for progressive muscular atrophy. If we bear in mind its sudden or almost sudden and complete origin; the absence as a rule of fibrillary tremors (only two cases which presented these symptoms having been reported); that the paralysis precedes the atrophy, and retrocedes after the first general attack; that electric irritability is primarily lost; and that the atrophy involves the muscles of one or more (usually two) extremities, there need be no error made in diagnosis. Anæsthesia, incontinence, and paralysis of the sphincter ani prevent it from being confounded with general myelitis, these symptoms belonging to the latter in addition to the loss of power and atrophy. *Spinal congestion* may sometimes give rise to some of the symptoms, and Cartwig<sup>4</sup> presented a case which he called "intermittent," somewhat resembling the lighter form of true antero-spinal paralysis.

A sugar-baker, aged 23, who was exposed to great heat and sudden changes of temperature while very lightly clothed, had suffered in his eighteenth year for four or five weeks from an attack of tertian ague, from which he recovered. One day he perceived a numbness in his legs, which rapidly attacked his arms also, and finally led to complete para-

<sup>1</sup> Thèse de Paris, 1872.

<sup>2</sup> Archives de Physiol., norm. et path., tome v., 1873.

<sup>4</sup> Centralblatt f. d. med. wis., June 15, 1870.

<sup>3</sup> Op. cit.

lysis of the muscles of the neck. Speech, deglutition, and respiration were somewhat impeded; the muscles of the eye were unaffected, as were also the alvine and urinary excretions, and sensation. After twenty-four hours there was a remission of the symptoms; first the neck began to become movable, then the fingers, arms, body, and finally the legs. All this took place in half an hour, and was followed by an increase of perspiration. During the next twenty-four hours the patient remained free from paralysis, but was dull; after which, the above-described symptoms returned. The brain was always free; the cervical portion, especially the upper, was not always equally affected; the movements of the neck were often free; and difficulty in deglutition and respiration, inequality of the pupils, and myosis, were frequently present. The phrenic nerve was always unaffected. When there was not complete paralysis, the affected limbs were generally stiff, and there was contraction of the predominating groups of muscles; when complete paralysis was present, the muscles were soft and flabby. Electro-muscular irritability was almost completely absent during the paralysis, and the violence of the muscles varied. Under the use of quinine, the patient's condition was on several occasions quickly improved, but he was not cured. He was under observation for more than six months. The author believes that the case was one of masked intermittent, and that the phenomena were due to hyperæmia of the cord and occasional increase of serous exudation.

In spinal congestion there are no deformities, no atrophy, and nearly always vesical trouble and constipation.

*Acute ascending paralysis* resembles very closely certain forms of the disease under consideration. In one remarkable case reported by Desjérine,<sup>1</sup> no morbid appearances were found after death. A man entered the hospital suffering from undefined pain in the lower limbs, and two days after became paraplegic without any loss of sensibility. The paralysis rapidly succeeded, and, after four days, he died; no trace of disease after paralysis of the respiratory muscles could be found except dilated vessels.

Seguin considers that this involvement of the respiratory muscles is a diagnostic sign.

**Prognosis.**—Antero-spinal paralysis is not a disease which is rapidly fatal, and many cases recover within a short time after the beginning of the attack. I am not disposed to think that the lesion is an ascending one; but rather that, if it progresses at all, it involves the posterior and lateral parts of the cord in the majority of cases, and does not spread longitudinally. This is probably the condition of affairs in the case of S. W. Should the paralyzed muscles become atrophied to such an extent that deformities result, I think that there is very little hope for the patient. If, however, the muscles can be made to respond to the galvanic current, we should never be discouraged.

Of the cases reported by Duchenne, Meyer, Bernhardt, Seguin and others, I find that of 16 cases there were but 2 deaths. In one observation there was improvement in six months, in another in four, and in others two,

<sup>1</sup> Archives de Physiol., etc., June, 1876.

three, eleven, and twelve. In two cases the patients were cured, and in several there was progressive unfavorable advancement.

**Treatment.**—In electricity we possess a remedy of the greatest value. I have already called attention to its use in the infantile form of the disease, so there is no need for going into details. It is well to use both the galvanic and faradic currents, and in the acute form of the trouble we should begin with counter-irritation of the spine as early as possible, and for this purpose may employ blisters or the actual cautery.

Ergot and belladonna in rather full doses should be employed in conjunction therewith. Seguin recommends leeching and dry cups, which are both excellent.

Should the pain be severe, we may use morphine by means of the hypodermic syringe; or spinal galvanization. The after treatment should be with the galvanic current.

The use of warm applications, such as have been spoken of as of benefit in the infantile variety, are worthy of trial.



## CHAPTER X.

## DISEASES OF THE SPINAL CORD (CONTINUED).

## PROGRESSIVE MUSCULAR ATROPHY.

**Synonyms.**—Wasting palsy; Cruveilhier's paralysis; Progressive muskelatrophie; Progressive muskellähmung.

**Definition.**—This is an essentially progressive atrophy of certain groups of muscles. It is not *preceded* by any paralysis, but followed by loss of power, and terminates usually by involvement of the respiratory nerve-centres.

Cooke,<sup>1</sup> in 1795, directed attention to a condition he called "*anomalous hemiplegia*," which was clearly progressive muscular atrophy, and his was probably the first recorded case. Bell,<sup>2</sup> Abercrombie,<sup>3</sup> and Darwell<sup>4</sup> each published cases which were undoubtedly of this kind; and, in 1836, Mayo<sup>5</sup> related two cases. It was not, however, till 1849, when Duchenne de Boulogne<sup>6</sup> presented a memoir to the Institute of France, entitled "*Atrophie musculaire avec transformation graisseuse*," that the present disease was recognized. In 1853, Cruveilhier<sup>7</sup> described some cases in which the atrophy was general, all the voluntary muscles being affected. In 1850–1861, Aran,<sup>8</sup> Duchenne,<sup>9</sup> and Eisenmann<sup>10</sup> brought forward additional facts, and the latter agreed with Cruveilhier that the "nerves or nervous centres are at fault anterior to the muscles, and that the atrophy of the latter is a secondary process." Since that time we are indebted to Roberts<sup>11</sup> and Friedreich<sup>12</sup> for most clear and instructive descriptions.

**Symptoms.**—The appearance and progress of the disease are most gradual. The affected individual may first notice a slight weakness in one of the upper extremities. Perhaps the first indication of trouble which suggests to the patient the commencement of the

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<sup>1</sup> Cooke on Palsy, p. 31, 1822.

<sup>2</sup> The Nervous System of the Human Body, London, 1830.

<sup>3</sup> On the Brain and Spinal Cord, p. 419, Edin., 1828.

<sup>4</sup> Lond. Med. Gaz., vol. vii., p. 201.

<sup>5</sup> Outlines of Human Pathology, p. 117, London, 1836.

<sup>6</sup> Mémoires de l'Acad. des Sciences, 1849.

<sup>7</sup> Archives Gén. de Méd., May, 1853.

<sup>8</sup> Ibid., Sept., 1850.

<sup>9</sup> De l'Electrisation localisée, Paris, 1855–61.

<sup>10</sup> Canstatt's Jahresbericht, 1859.

<sup>11</sup> An Essay on Wasting Palsy, London, 1858.

<sup>12</sup> Ueber progressive muskelatrophie, etc., Berlin, 1873.

disease, is when the act of writing is attempted. According to Roberts, the disease begins, in two-thirds of the cases, in the upper extremities, and the muscles of the hands are the first to suffer loss of function. Very often several muscles are affected together, and they soon become agitated by what are known as *fibrillary contractions*, or, as they have been called, *vermicular contractions*, which in their nature are probably a divided reflex excitation. The subcutaneous contraction of muscular filaments suggests the appearance of worms crawling beneath the skin, and there is sometimes a species of *muscular shivering*. These fibrillary contractions may be excited by sharply striking the muscles with a ruler on the hand, and they sometimes follow the passage of the galvanic current through a nerve-trunk. As I have said, the hand may be affected first, and there may be extensive wasting here before other parts are attacked. The muscles of the palm of the hands, when atrophied, give to that member a most unsightly appearance. The bones stand out in strong relief, and the thenar and hypothenar eminences are flattened, and the flexor tendons are prominent, and increase the deformity. With this there is contraction of the flexors, and the hand resembles more the claw (Fig. 43)

(Fig. 43).



"main en griffe" (Duchenne.)

of an animal than anything else, so that it has been called "main en griffe." The back of the hand also presents a most skeleton-like aspect, the extensors, the interosseal muscles, and sometimes the adductors of the thumb having been reduced in size. The forearm and arm are next to follow, and rapidly lose their normal conformation. The deltoid and serrati muscles may be involved, while those of the arm proper may occasionally be passed over. The head of the humerus and angle of the scapula are quite distinct, and this bone may be drawn out of place by the healthy muscles, this being the rule when the serratus magnus is the seat of atrophy. The angle of the scapula is drawn upwards and inwards, and stands out from the trunk. It is rare to find symmetrical atrophy, and in the majority of cases I have seen there has been a great difference in the invasion of muscles on the two sides. The right upper extremity appears to be the favorite seat of the atrophy, while the lower extremities are quite rarely affected, and in the proportion of 1 to 12 to the upper extremities. The muscles of the face and head are sometimes the seat of atrophy, but this is unusual, though muscles may occasionally be so extremely wasted that there is no appearance of intelligence whatever. The eyes, of course, being unaffected, are the only agents of expression. There may be atrophy of the tongue and buccal muscles, with disturbances of speech and drooling of saliva, and in such cases death usually follows in a very short time. Sometimes the muscles of the neck do not escape the extension of

the disease, and the chin falls forwards and downwards. The last muscles involved are generally those concerned in respiration; and not only are the intercostals the subjects of such a change, but the diaphragm is finally paralyzed, so that the action of the lungs is interfered with, and ultimately the patient is literally asphyxiated. Subsequent to atrophy, a loss of power takes place. The affected muscles preserve for a long time their electric contractility; but this is finally lost as they decrease in size, and loss of power increases till finally the patient becomes helpless. Duchenne is of the opinion that the loss of *voluntary* muscular contractility is rather the consequence of atrophy or textural alteration than of paralysis, *i. e.*, loss of motor innervation ("C'est-a-dire du défaut d'action nerveuse motrice"). Tactile sensibility is, however, rarely blunted. One of the earliest symptoms of progressive muscular atrophy is the presence of dull pains in the affected limbs, and this has led very frequently to a mistake in diagnosis, the condition being often considered rheumatic. In one case sent to me by Dr. E. G. Loring, I found that the atrophied muscles were the deltoid, serratus magnus, and biceps, but none of the lower muscles of the forearm were attacked. The man had consulted another physician, who considered the case one of chronic rheumatism, and prescribed liniments and alkalies. The patient was an upholsterer, and had been obliged to use his right arm to a great extent, especially in hammering on cornices, and putting up decorations which were above his head. He had had violent pain in the shoulder for some months, and subsequently the atrophy began in the deltoids. When I saw him the head of the humerus was prominent, and there were fibrillary contractions in some of the muscles of the back. When the upper extremity is affected, it will be found that when the forearm is flexed the belly of the biceps will be often found to be reduced to the size of a small ball. The progress of the disease is marked by the occurrence of well-marked intermissions, and a year or two may often pass without any extension, while at the end of that time a fresh start is taken, and two or more of these stationary periods are not uncommon in the course of the malady. The ordinary tendency of the affection is however progressive; and although, as I have said, the disease may pursue the most eccentric course, attacking groups of muscles here and there, it will involve ultimately a very great number, and finally those supplied by the lower cranial nerves, unless it be checked by proper treatment.

I may illustrate the symptomatology of progressive muscular atrophy by a case which ran a somewhat irregular course by attacking the muscles of the lower extremities:—

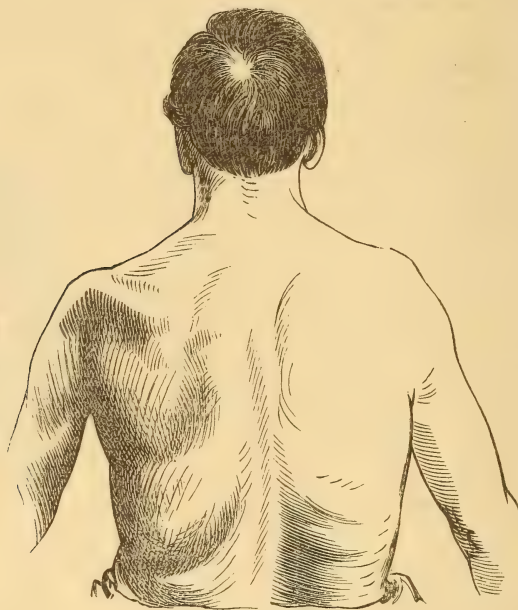
J. F. H., 31 years old; U. S.; engineer. Twenty-one months ago the patient, after exposure, developed what he says was articular rheumatism, which chiefly affected the legs. On recovery he noticed that the right leg "began to grow smaller at the calf," and that afterwards his left thigh became smaller. His pains continued at intervals, and were increased by damp weather.

*Present Condition.*—The muscles of the anterior part of legs and thighs



are much wasted, the abductors of thighs and the recti femoris on both sides being notably so. The knees seem very large, and the condyles of the femur are felt to be superficial and covered tightly by the skin. There is no loss of sensation, and electric irritability appears to be very generally preserved, except in the recti femoris. The glutei muscles have suffered to some extent on both sides. He has severe pain at night, which runs down the legs, and "seems to be deep." There is impaired motor power, and he finds that walking is difficult. He does not experience any urinary trouble, and his bowels are not constipated. There is no loss of co-ordinating power, no constricting band, no history of any kind of acute myelitis. The muscles on the outer side of the thigh are the seat of fibrillary contractions, which occur sometimes when he makes a voluntary effort. There was at this time no atrophy of any of the muscles of the upper extremities, but when I saw him some months subsequently there was commencing atrophy of the muscles of the right hand. In the paralyzed

Fig. 45.



Atrophy of the Left Shoulder.

muscles the temperature is much lowered, and this is a constant feature of the disease.

Jaccoud<sup>1</sup> and others have called attention to a temperature change, which they call "refroidissement variable," in which there are times when the temperature may fall several degrees, and this seems to be the result of a paroxysmal ischæmia of the tissues. The pupillary condition is an interesting feature of the disease, the dilators sometimes being paralyzed, so that the pupils are widely or unequally dilated.

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1 Op. cit., p. 326.

It is the rule, in these cases, to discover certain trophic changes affecting the skin and its appendages, so we quite commonly find diseases of the nails, eruptions, and other cutaneous lesions; but a patient now under treatment presents something in addition to these. It has been found that he sweats profusely upon the right side of the body, which is more atrophied than the left, while the left side is quite dry.

By careful experimentation I have found that when ammonia is held to his nose the right eye almost immediately becomes suffused with tears, while the left remains almost entirely unaffected.

When salt is placed upon the tip of the tongue an abundant discharge of saliva from the right corner of the mouth occurs almost at once.

Dr. Claddek, my assistant at the Hospital, painted with cantharidal collodion two spots of the same size upon either side of the chest, and upon the normal side only very slight changes took place, while upon the right, or affected side, a blister was formed almost immediately, and it was very slow in healing.

In many cases the general health of the patient is unaffected in any way, and yet the atrophy may be of the most complete nature. I recently saw a patient thirty-eight years old, who had been a soldier in the regular army, and was exposed much to the elements. His illness has lasted but two years, yet in that short time nearly every voluntary muscle has undergone a great diminution in size, except those of the face. His respiration is labored, and he cannot stand without support. He is 5 ft. 8 in. in height, and his anterior dorsal curve is four inches in extent. In a line measured at level of nipples his chest girth is 26 inches; at inspiration there is a gain of two inches. The right arm at middle of biceps is  $6\frac{3}{4}$  inches in diameter; the left  $6\frac{1}{2}$  inches. All the bony prominences are distinct, the angles of the scapulæ approximate, and he is almost a skeleton in appearance. There is no loss of sensation:

The atrophy in this case was as great as that presented by Duchenne's patient,<sup>1</sup> Bonnard, in which the pectoral, trapezii, with the exception of their clavicular portion, great muscles of the back, biceps and anterior muscles of the left arm, supinators longii, had nearly entirely disappeared.

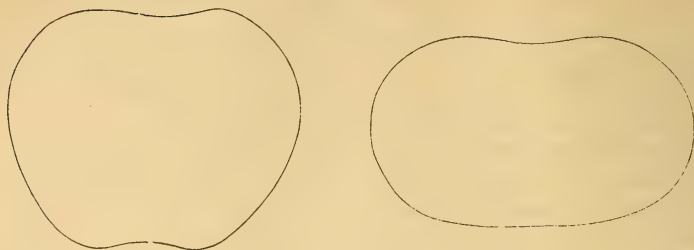
Duchenne alludes to the changes in conformation of the thorax when the intercostals or diaphragm are paralyzed, and presents two illustrations showing the perimeter of the chest in two patients affected with atrophy. These are presented in the accompanying illustrations. Thoracic troubles, such as bronchitis, are not uncommon as a result of impaired lung action.

**Causes.**—These may be enumerated as *heredity*, which is found to enter conspicuously into the etiology of progressive muscular atrophy, *exposure*, the *over-use* of particular groups of muscles, *injury of the spinal cord*, and sometimes *syphilis* and the *zymotic diseases*. As to the hereditary influence which favors its development, Friedreich<sup>2</sup> reports several cases, which go to show that this disease, more than all others, commonly

<sup>1</sup> Op. cit., 3rd ed., p. 500.

<sup>2</sup> Op. cit.

Fig. 44.



(Duchenne.)

appears in several generations of the same family. I have seen one case where it could be traced for three generations back, and in another, which I will presently detail, there were uncles and aunts affected. Eichert,<sup>1</sup> in a very valuable article, gives the family history of one case. In a genealogical table he traced the disease back six generations, and representatives of these generations are still living. Seven cases are related by him. In two of the cases the parents have escaped, while the children have suffered. It is unnecessary to pursue this matter further; but I am firmly convinced that there is no other disease, except perhaps it may be phthisis pulmonalis, which is transmitted so frequently as this terrible malady. Exposure to damp, neglect to change wet clothing, and like imprudences, are exciting causes in many cases. Neuralgic pains are very prominent in such cases, and the onset of the disease is rather precipitate. Mechanics of all kinds, who are in the habit of using some muscles much more than others, are frequently the victims of the disease, and the muscles which have been over-used are affected before the others. I have seen the same limited atrophy in a cigar-maker and in a compositor, who used certain groups of muscles almost constantly. Roberts has dwelt upon the connection between injury of the spinal cord and the disease under consideration; and Valentiner,<sup>2</sup> Bergmann,<sup>3</sup> and Thudicum have all called attention to the appearance of the disease some time after the receipt of an injury. Roberts reports a case in which atrophy of the ball of the right thumb, and subsequent complication of the respiratory muscles, and death followed a slight injury received six months before. The other cases are none the less interesting, and go to prove the importance of recognizing such causes. As to age and sex, it has been found that progressive muscular atrophy is not confined to any period of life, but the bulk of cases occur after puberty. Of 88 cases reported by Roberts, 1 was only 2 years old, and another 69. Of the 28 cases I have seen, the atrophy began in 2 between the 5th and 10th years; in 5, between the 10th and 15th; in 18, between the 20th and the 30th; and in 3 after the 30th. Of these, 23 were men, and but 5 women. This

<sup>1</sup> Prag. Viert., 1855.<sup>2</sup> Berliner Klin. Wochenschrift, Oct. 20, 1874.<sup>3</sup> Petersburg Med. Zeitsch., 1864.



seems to be the rule, and Roberts states that six men are affected to every woman, and he considers this due to the exposure and external violence to which males are subjected.

**Morbid Anatomy and Pathology.**—The disputed point in regard to the pathology seems to be whether it is a primary peripheral condition, or whether it is a central affection in which the trophic cells are affected. The advocates of the first theory call attention to the fact that muscular atrophy occurs independent of any loss of the muscular function, and believe it to be purely a local degeneration. The authorities I have spoken of, in alluding to the early history of the disease, all believed in this intra-muscular origin; but lately there have been so many proofs of its central origin brought forward, that the former theory has been abandoned. This difference of opinion seems to exist in regard to the form of central lesion. The majority of observers are agreed that there is an affection of the anterior horns; and that the change is one that affects the trophic cells of Duchenne and Westphal, and the fibres which connect with sympathetic ganglia.

To Lockhart Clarke,<sup>1</sup> who has so often decided questions regarding the pathology of nervous disease, belongs the credit of having discovered the central origin of this disease. He found atrophy of the anterior gray horns, and since his original observations many other observers have come forward to endorse his views. Von Recklinghausen and Dumenil<sup>2</sup> disagree, however, with this view, and the microscopical examination made by the former was unattended with any discovery of morbid appearances.

Jaccoud has collected six cases in which fatty degeneration of the sympathetic had taken place, and one of them was observed by this author himself. Not only was there fibro-fatty degeneration of the sympathetic nerve, but there was atrophy of the anterior roots. The view held by Jaccoud is that the trophic filaments of the sympathetic which preside over nutrition do not perform their duty, and that the affection of a mixed nerve, which contains motor, sensor, and trophic filaments, at a point where they are intimately mixed, must result in a perversion of all their functions; but if the separate filaments be attacked at a point before they become blended, there may be independent loss of function of either one.<sup>3</sup>

Charcot and Gombault<sup>4</sup> have described the following interesting *post-mortem* appearances witnessed in a recent case:—

<sup>1</sup> Brit. and For. Med.-Chir. Review, vol. xxx., 1862.

<sup>2</sup> Gaz. Hebdom., 1867.

<sup>3</sup> The localization of well-defined lesions in this disease is sometimes made before death and verified afterwards. Prevost and Cotard (Archives de Physiol., Sept., 1874) present such a case. There was atrophy of the right thenar eminence, with atrophy of the right anterior root of the eighth pair of cervical nerves, slightly marked atrophy of the right anterior root of the seventh cervical nerves, and atrophy of the gray matter of the anterior horn at this level of about an inch in extent.

<sup>4</sup> Archives de Physiol., 1875, No. 5, abstr. Phil. Med. Times.

"No change in hemisphere, cerebellum, pons, or medulla oblongata in these nerves. The gray substance of the cervical and dorsal medulla spinalis was greatly altered from the lower portion of the cervical enlargement down, gradually decreasing downwards and outwards. The nerve-cells and nerve-fibres of the anterior gray cornua had disappeared; the capillary vessels were greatly developed; the parietes of the smaller and larger vessels were thickened. The lumbar portion of the cord and the lateral columns were normal. In the cervical and dorsal region, the portions of the cord near the merging external roots were sclerosed; the change being proportionate to the intensity of that which had taken place in the gray cornua. The few ganglion-cells present were very much diminished in size, without processes, more rich in pigment than normal, but still containing nuclei and nucleoli. The anterior roots of the cervical region were atrophic; empty sheaths, frequently containing large nuclei, appeared in place of the normal fibrillar contents. The posterior roots seemed normal.

"As to the peripheral nerves, one phrenic and several intercostal nerves were examined; more than two-thirds of the nerve-tubules (in hardened sections) were wanting, by a process similar, as it would appear, to that induced by an external wound. The muscles about the shoulder and the upper extremities were for the most part atrophic; there seemed to be a peculiar atrophy of the primitive fasciculi, without any marked alteration in the fibrils, and without any excessive development of the interfibrillar fatty tissue."

The changes discovered by Clarke<sup>1</sup> were in the gray matter. There was a granular deposit about the vessels, and corpora amylacea about the central canal. Lesions of the anterior nerve-roots were found, and in the cervical region there seemed to be more distinct appearances than at any other point, where it will be remembered there may be found sympathetic as well as motor and sensor fibres.

The muscles present distinct evidences of fatty degeneration and fatty substitution. They appear to the naked eye as wasted bands which contain lines of fat. The appearance of healthy muscles of good contour in juxtaposition with others which have undergone atrophy is very peculiar, and it is difficult to realize that the disease can involve such isolated tracts. The muscles of the lower extremities may have undergone general fatty degeneration. A specimen prepared by my friend Dr. Weisse, of the Medical department of the N. Y. University, shows very beautifully this condition of affairs. Fatty substitution has gone on to such an extent that there is no appearance of muscular fibre to be seen, but every muscle exists as a distinct band of adipose tissue. Atrophied muscles have been examined by Meryon,<sup>2</sup> Galliet,<sup>3</sup> and others, and their descriptions of appearances agree very closely. The muscular structure suffers a complete change, the striæ disappearing and the sarcolemma undergoing a granular change. Fox<sup>4</sup> divides the secondary changes into the fatty degeneration

<sup>1</sup> Med. Chir. Trans., 1851, 1856.

<sup>2</sup> Ibid., 1866.

<sup>3</sup> Archives Gén., vol. i., 5me série, 1853, p. 584.

<sup>4</sup> Op. cit., p. 266, et seq.

which takes place *inside* of the sarcolemma, and as an *interstitial* deposit. These he calls the *parenchymatous* and the *interstitial*. Sometimes, as observed by Robin, the atrophy may take place as a fibrous degeneration, or species of muscular sclerosis. Some muscles appear as fibrous cords of a white color, while others may be found which have undergone the fatty degeneration just described.

An instructive case in which very striking appearances were presented was observed by Dr. Janeway, whose observations are recorded below:—

M. G., aged 62 years, widow; admitted to hospital December 16th, 1873. Right hand: the muscles of ball of thumb are very much atrophied, and she is unable to move it; there is also slight rigidity of the joints of the thumb.

Dorsal interossei are very much wasted; there is slight power of flexion and extension of fingers, especially little fingers, and there is also a slight movement at the wrist.

Sensibility good except in index finger, and here it is decidedly diminished. She can raise her arm to her head and place it in any position. Hands seem cold.

Left hand is not so much affected; the muscles of ball of thumb are partially wasted. The abductor opponens and outer head of flexor brevis are almost gone; the inner head of flexor brevis and abductor partially, and capable of acting to a slight extent. Has slight power of ab- and adduction of fingers, especially the little finger, most on the ulnar side, and decreasing toward the radial; has slight power of extension over fingers, none over thumb, but flexion power is more marked. Has no power of extension, but considerable of flexion at the wrist.

Dynamometer L. H. 28. Sensibility normal; hands cold. The muscles that are capable of acting respond to the induced current very well.

July 9. Complains of dizziness and nausea

17th. Dizziness still. Her hands are in same condition. She experiences some difficulty in walking, and moves with her body "sloping over" She cannot use her hands, and when she attempts to do anything, they drop, and she cannot raise them. The muscles that remain unaffected respond well to electricity. She still vomits at times after eating.

August 3 Is quite weak; has chilly sensations.

4th. Had a severe fever last night; temperature 104°; passed feces in bed, and did not know it; to-day temperature is almost normal; is quite apathetic.

5th. Has chilly sensations; complains of no pain; arms and jaws tremble; temp. 102°.

2 P. M. Temp. 102°.

6th. She is very much worse; mucous râles heard all over chest; respiration accelerated; temp. high; pulse very feeble; pupils normal; bowels moved once to-day; swallows with great difficulty.

2. P. M. She sank gradually and died at 12.45 P. M.

*Post-mortem, held twenty-seven hours after death.*—Rigor mortis moderately well marked. Nearly all the muscles of the hands are atrophied, especially the dorsal interossei and the propria muscles of the thumb; the change is nearly symmetrical in both hands. The forearms are extremely wasted, both on the flexor and extensor surfaces. There is no marked



wasting in the arms, the shoulders are well rounded ; both pectoral regions appear wasted ; there is no marked wasting in the lower extremities, unless it be in the adductor region of both thighs. Incisions made into the pectoral muscles, show well-colored fibres also in the deltoid, biceps, and triceps.

The extensors of the forearms are of whitish-yellow color, being nearly as pale as the skin.

The flexors of right hand are very much wasted, but not so much as the extensors. The flexors of the left side are small, but seem in good condition.

The muscles of the right thenar eminence show extreme degeneration. In left thenar eminence the inner head of flexor brevis and adductor are red and large ; the external is white, as on the other side. The adductors of thighs are small, but well-colored.

The quadriceps extensor femoris is of good color.

The anterior tibial muscles are of good color.

Heart: Valves are normal, muscular substance soft, and yellowish-gray. The diaphragm is not atrophied.

Brain: Convolutions and corpora striata appear normal. There is some atheroma of the carotid and basilar arteries.

The substance of the cord and brain is quite soft. The viscera are normal, except the kidneys, and these are granular ; their pyramids are small, and they contain small cysts.

**Diagnosis.**—Progressive muscular atrophy may be mistaken for several conditions of a paralytic nature, among these *lead paralysis*, *antero-lateral sclerosis*, *partial paralysis* from traumatism, and infantile or adult paralysis.

For an illustration of the first of these I do not think I can do better than mention a case in which there appeared to be lead paralysis, but which subsequently turned out to be progressive muscular atrophy.

Several months ago, Mr. N., a Cuban gentleman, came to me with a letter from his medical adviser, Dr. Findlay, of Havana. The doctor's history of the patient is as follows: "Mr. N., about eighteen months ago, began to experience a tremor in the fingers and wrist of the right hand, together with muscular debility, which caused some inconvenience in writing, and in carrying food to his mouth, as well as in other movements of the hand. Having on a single occasion submitted to local faradization of the arm (some ten months ago), the tremor was much subdued, and, as was thought, the fingers and wrist were strengthened. It was not, however, until four months ago that the patient returned to put himself under a regular course of treatment.

"*Condition of the patient in July, 1876.*—General health good ; no signs of cachexia ; no antecedents of specific taint ; no lead poisoning. Suffered on two or three occasions, at some years' interval, rheumatic pains and neuralgia in the arm and shoulder of the left side, but never in the right side, which is the one now affected. The outer appearance of the right arm showed but little muscular atrophy ; the tremor was inconsiderable ; the patient could close the hand tightly, but not well grasp larger objects, such as a tumbler, owing to incapacity to maintain the first

phalanx of the third, fourth, and fifth fingers extended. The wrist was inclined to drop forwards (in flexion) and outwards.

"On inspection it was found that the common extensor of the fingers was considerably weakened, most so in the portion attached to the ring-finger, the weakness being manifested both to voluntary and to electrical contractility. The same condition existed also, though a little less, in the extensor of the little finger, and in the radial extensors. The contractility was not totally absent, but would vary in degree without apparent cause. The disease continued to progress (notwithstanding treatment), the portions of the common extensors losing all excitability to my small Gaiffe's battery, and the extensors of the thumb being also implicated.

"The left arm was now examined, and although the patient did not notice any weakness in the hand, yet some deficiency of electric contractility was observed in the common extensor, especially in the extensor of the ring-finger. The constant current was now used for six weeks without much benefit. The extensor carpi ulnaris is now becoming also affected. The patient, however, finds that he can write and perform various acts with the right hand better than before. Within the last week he complains of some pain along the back of the left forearm when he has been holding an object in the air, and feels an inclination to relax his grasp."

The doctor also gave a history of hereditary trouble, which was probably in one case (the patient's uncle) progressive muscular atrophy.

I carefully examined the patient, and found that the right arm was that most affected.

*Motor power.*—The power of extension of the muscles of the right forearm was lost completely, and on the left side the power of extension of the two middle fingers was to some degree impaired. Flexion was perfect.

*Atrophy.*—The following muscles were more or less affected and reduced in size. Right forearm: Extensor communis digitorum; extensor minimi digiti; extensor carpi radialis; extensor longis pollicis; extensor carpi ulnaris; extensor communis of the left.

*Sensation.*—Slightly impaired on the right side. The teeth of the æsthesiometer were separated by a space of about ten centimetres before two points could be appreciated. This loss was not so great on the under surface of the forearm. There was no history of recent pain either constant or neuralgic, nor were there any dysæsthetic sensations.

No fibrillary contractions were observed. There was a slight tremor in the right hand when voluntary movements were made. Electric contractility to a very slight degree was observed in the extensor communis digitorum when a strong faradic current was applied. The galvanic current also seemed to have some influence upon the weakened muscles. The fingers were covered by small flakes of skin, and the nails were crenated, irregular, and evidently badly nourished. This trophic defect disappeared under the use of the galvanic current.

*Diagnosis.*—In the order I name them I proceeded to dispose of lead pæresis, amyotrophic sclerosis, cerebral paralysis, traumatic paralysis, and progressive muscular atrophy.

That it might be lead pæresis seemed reasonable at first, because of the loss of electric contractility, the seat of the paralysis, etc.; but when I bore in mind that the trouble was one-sided at first, that there was a subsequent invasion of the muscles of the other arm, that sensibility was also

impaired, and that the patient used neither hair-dye nor drank impure water, nor was exposed to the dangers of lead poisoning of any kind, I was forced to abandon this idea. A species of spastic contraction drew down the fingers of the right hand and there was some cumulative tremor, such as characterizes sclerosis (expressed by a gradually increased tremor, aggravated by will control, and terminating in a species of spasm). This at first led me to suppose that there might be some degeneration of the lateral columns, but as the tremor disappeared and there were no other symptoms of such degeneration, and especially as there was gradual atrophy and muscular paralysis, I dismissed this possibility. The loss of electric contractility, and the limited field of the paralysis, excluded cerebral paralysis; and the fact that the patient had never received an injury, and that the affection was beginning to affect the opposite group, negated the theory of traumatic paralysis. All that was left was the diagnosis of progressive muscular atrophy; and the subsequent appearance of fibrillary contractions made me quite sure of my decision. The slow progress of the trouble and its site were, however, doubtful points. The individual had not exercised any particular member, and as he was a man of leisure, there was no trade or occupation in which constant use of the hands or excessive labor was required that could account for its origin. The hands preserved their contour; there was no atrophy; no prominent thenar eminences; nothing suggestive of the *main en griffe*. None of the muscles of the back were affected, and the deltoids were of good volume and power. The fact that others in his family had suffered, that the disease began on one side and extended to the other, that fibrillary contractions were present, that subsequently I was enabled to get slight, and afterwards stronger contractions of the paralyzed and atrophied muscles, determined me in my diagnosis of this anomalous case. I call it anomalous, because I have been taught, and my own experience convinces me, that this is a very rare seat of progressive muscular atrophy. Protean as is the malady, I have not seen paralysis of the extensors, as a primary symptom, in any one of the twenty-eight cases of the affection I have met with from time to time.

In lead paresis the invasion is rapid, the paralysis the same, and the atrophy is secondary, which is not the case in the wasting palsy. There is sometimes the lead line or lead colic, and electric contractility is impaired from the first. From traumatic paralysis it can be diagnosed by the irregularity in situation of the muscles atrophied. In traumatic paralysis we may look for atrophy of groups of muscles which are supported by a common trunk, as well as loss of electric contractility and *secondary* atrophy.

The diagnosis from some forms of adult and infantile paralysis is not so easy. In fact Duchenne believed the pathology of the two affections to be nearly the same. The sudden origin of the infantile cases of course precludes any mistake in the majority of cases, but in adult cases even after the disease has existed for some time.

In such cases the paralysis and atrophy may co-exist to a disproportionate degree. If it is possible, however, to ascertain the early occurrence of paralysis, and if the loss of muscular substance be rather general, no mistake need be made.



**Prognosis.**—Occasionally the malady may be arrested or cured entirely, and this fact seems almost incredible when we bear in mind its organic character. The duration of the disease is variable. Some of these patients recover, while in other cases it runs its course in from five to twenty years, the atrophy meanwhile involving fresh groups of muscles with more or less rapidity.

In a case shown at my clinic, the disease had lasted for two years, and the atrophy had involved nearly all the muscles of the upper part of the body. In another patient I have recently seen, the disease has progressed very little during the last ten or twelve years.

I have succeeded in arresting the disease in ten cases, and think that, when there is the least muscular response to electricity, there is still a chance for improvement, if not complete relief. This is, of course, in proportion to the extent of invasion. If the atrophy be confined to the muscles of one forearm, there need be no reason to give a bad prognosis. The majority of cases, however, go on to an unfavorable termination, and perhaps one reason is, that patients delay so long to seek medical advice, considering their disease to be rheumatism, and amenable to domestic treatment. When the diaphragm or the intercostales are invaded, the prognosis is as bad as it well can be.

Roberts<sup>1</sup> thinks that the prognosis is bad when hereditary predisposition can be traced, or when the upper and lower extremities are both implicated.

**Treatment.**—I know of no other remedies than those which are local (except when a syphilitic taint is suspected). Electricity is one of these; muscular rest is the second when the affection has followed over-use of certain muscles.

The galvanic current from not less than twenty cells should be used, one electrode being placed over the nucha, and the other in the supraclavicular space. Applications of ten minutes every day cannot fail to do good. In addition to this, the faradic current should be employed for the muscles themselves, making each muscle contract several times, and then allowing it to rest, and repeating the operation some minutes afterwards. Violent electrization, I am convinced, fatigues these crippled muscles, and does more harm than good.

Duchenne gives the following directions for the use of the induction current: "Place the wet electrodes, so that they are as near together as possible upon the surface of each of the diseased muscles, using an induction current of greater or less tension, so that all the anatomical elements of the muscle shall be excited. Excite the muscles generally and moderately and apply an intermitted current. Faradize only the atrophied muscles which still respond to electric excitation, among the latter, faradize by preference those which enter most frequently and usefully into important muscular movements. End each seance by the slow faradiza-

<sup>1</sup> Art. Wasting Palsy, Reynolds's System of Medicine, American Edition, vol. i., p. 796.

tion of the more important muscles among those threatened by the invasion of atrophy."

Vivian-Poore and Fagge<sup>1</sup> have had wonderful success with this agent, and have cured a number of apparently hopeless cases. I have been induced to try the "rubber muscle," as arranged for lead palsy. This forms an admirable means for support of the hands, should the extensors be affected, as was the case in the history I have just related. In every case it is well to insure perfect rest, if possible, for all affected muscles. If the muscles of the shoulder be so atrophied as to allow the arm to drop, it is well to arrange some contrivance to sustain its weight, and relieve the strain upon the affected organs. Sulphur baths and mineral waters have been recommended, and in some hands have been successful.

### PARTIAL FACIAL ATROPHY.

**Synonyms.**—Trophic neurosis of the face (Romberg); Laminar aplasia (Lande); Unilateral progressive atrophy of the face (Eulenburg<sup>2</sup>).

**Definition.**—A disease of a trophic nature, involving usually one side of the face, beginning with discoloration and cutaneous changes, and ending in loss of tissue of underlying cellular tissue and bone, not accompanied by loss of motor power or sensibility.

The disease was, according to Eulenburg, first described by Parry<sup>3</sup> in 1825, and afterwards described by Bergson<sup>4</sup> in 1837.

It subsequently received attention from Romberg,<sup>5</sup> Lande,<sup>6</sup> Samuel,<sup>7</sup> Eulenburg,<sup>8</sup> Frémy,<sup>9</sup> Moore<sup>10</sup> and others, who described many cases. Eleven cases are reported by Lande alone. The first American case was presented by Dr. Draper<sup>11</sup> before the New York Society of Neurology, Dec. 20, 1875, and other cases have been brought forward since by Seguin, Robinson, Bannister and others.<sup>12</sup>

A photograph of Dr. Draper's case is presented below.

The patient, who was a stout, hearty Irish girl, aged 18, and without any hereditary predisposition, presented herself, with the following history: About two years ago the muscles under the body of the lower jaw of the left side began to diminish in size, and after a few months there

<sup>1</sup> London Practitioner, December, 1868.

<sup>2</sup> Ziemssen's Cyclopædia, p. 57, vol. xiv.

<sup>3</sup> Quoted in Eulenburg's article.

<sup>4</sup> De prosopodysmorphia sive nova atrophie facialis specie, Berlin, 1837.

<sup>5</sup> Klinische Ergebnisse, 1846, and Klinische Wahrnehmung, etc., 1851.

<sup>6</sup> Essai sur l'aplasie lamineuse de la face en particulier these de Paris, 1869.

<sup>7</sup> Die trophischen Nerven, Leipzig, 1860.

<sup>8</sup> Wiener Med. Woch. und Lehrbuch der functionellen Nervenkrankheiten, 1871.

<sup>9</sup> Etude critique de la trophonévrose, Paris, 1873.

<sup>10</sup> Dublin Quarterly Journal, 1852.

<sup>11</sup> Am. Psychological Journal, Feb., 1876. Also consult recent cases in Bull. de la Soc. de Chirurgie, vol. 2, 1876. Gaz. Hebdomadaire, No. 13, p. 196, 1876. Br. Med. Journal, Aug., 1876.

<sup>12</sup> Journal of Nervous and Mental Diseases, 1876, vol. i.

was gradual extension of the atrophy, so that finally a district bounded by the symphysis of the lower jaw, angle of the nose, and middle of the upper lip in front, lower edge of zygoma above, and ramus of the inferior maxillary behind, became entirely affected. The skin is bound down to the periosteum of the lower jaw, and is shiny, tense and white. There never has been pain of any kind, but the only sensory alteration occurred in the beginning, when a slight itching was felt. There is no anæsthesia anywhere, not even in the tongue, one side of which is markedly atrophied. In the beginning there were occasional cramp-like pains about the insertion of the masseter muscles on the left side, but none on the other. There was slight paresis in some of the muscles involved.

Fig. 46.



Partial Facial Atrophy.

In twelve Continental cases collected by Draper, eight of whom were women and four men, the atrophy appeared in one at three years of age, and in another at twenty-two years of age. The beginning of the atrophy in these cases was not always the same. In two instances it began by pallor; in the others by red spots, next followed by loss of color; and finally there was a parchment-like appearance of the skin. Sensibility was not lowered in any instance, but in two there was itching, as in Draper's case. In one the disease was preceded by spasms of the masseter muscles; in six the tongue was atrophied; in one the tonsil; and in the rest the soft palate. In two cases there was deafness. In no case was there affection of the secretion of saliva; but in one there was diminished pulsation in the carotid of the affected side. In none were there indications of central disease. The cutaneous changes alluded to are peculiar, and a variety of trophic alterations may attend the disease; such, for instance, as falling out of the hair, or changes in color and the appearance of eczema. The sweat-glands do not seem to be involved, but the sebaceous secretion disappears upon the affected side. The atrophy is sometimes quite extensive, involving the bones, which, in some cases, have



been measured and found to be greatly reduced in size. Electric contractility of the muscles does not appear to be in the least diminished. The temperature of the affected side is generally lowered, but there is no diminution of sensibility. The left side appears to be the more common seat of the disease, and of the twelve cases already alluded to, but one was of the right half of the face.

**Causes.**—In some of the reported cases there was a history of previous intermittent fever, scarlatina (Hueter refers to whooping-cough as having had something to do with the genesis of this disease), and scrofula, and in one case there was a fall upon the head, but it is a question of great doubt whether these were concerned in the development of the atrophic condition. Courtet reports a case of right-sided facial atrophy in a subject who had been delivered with forceps. In this case the right pupil was the largest, which suggests the fact that there may have been some intracranial lesion. It seems, however, to be a disease which is more common between the tenth and the thirtieth year, and women are more often affected than men.

**Pathology.**—Undoubtedly this disorder is one of a trophic nature, and of central origin. The absence of motorial or sensorial disturbances makes this theory very plausible. If the lesion were of a peripheral character, it is highly probable that both sensation and motion would be affected, for I cannot conceive a diseased condition of trophic filaments alone when they are found in company with other sensor and motor filaments, as in a nerve-trunk which is diseased. This hypothesis seems more reasonable when it is borne in mind that the parts atrophied are supplied by other cranial nerves than the seventh. I therefore think that the theory of degeneration of the trophic cells of the bulb is a much more acceptable one than that held by Bergson and others. Eulenburg considers it to be essentially a lesion of the fifth pair, in which opinion he is sustained by Romberg, Samuels, Charcot, and Vulpian. Against this it may be urged that lesions of the fifth nerve of a trophic nature are generally followed by corneal changes, which, so far as I can learn, have never been witnessed in this disorder. Brunner is of the opinion that the condition is connected with a continued irritation of the cervical sympathetic upon the affected side.

**Diagnosis.**—Progressive muscular atrophy and facial paralysis seem to be the only diseases with which this may be confounded. Against the first it may be said that there are never the peculiar cutaneous changes of the disease under discussion—no dark spots, no falling out of the hair, no tightness of the skin; and moreover, this site of atrophy is very rare in progressive muscular atrophy. Facial paralysis is nearly always of sudden appearance, and the muscles lose their electric contractility.

**Prognosis.**—As far as I can learn no deaths have been reported, and no cures by drugs. From its progressive nature (and particularly if we concede it to be a central disease of a degenerative character) the prognosis must be bad, though two or three cases have been related, however,

in which there was an arrest of the atrophy without any treatment. In Belot's<sup>1</sup> case the disease became stationary after a year.

**Treatment.**—Electricity is indicated, but its use has only once been attended by slight improvement in the hands of Moore,<sup>2</sup> who reported a case which was benefited.

## PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

**Synonyms.**—Myosclerotic paralysis; sclérose musculaire progressive (Réquin); myosclerosis. Lipomatosis musculorum luxurians (Heller).

**Definition.**—A disease of infancy, expressed by increase of volume and hardness of certain muscles usually of the lower extremities, such increase being due to fatty substitution; by secondary atrophy and paresis and by conservation of cutaneous sensibility and the functions of the bowels and bladder.

Though first described by Sir Chas. Bell<sup>3</sup> in 1830, by two Italians, Coste<sup>4</sup> and Gioja in 1838, and subsequently by Meryon<sup>5</sup> in 1852, it was not until 1868 that the disease received much attention, when Duchenne<sup>6</sup> presented his collection of thirteen cases, with a critical analysis. At about the same time Meredith Clymer<sup>7</sup> was the first in this country to describe the condition. After him, Ingall and Webber,<sup>8</sup> Pepper,<sup>9</sup> Weir Mitchell,<sup>10</sup> and others, and among them Poore,<sup>11</sup> of New York, has fully discussed the subject, while numerous continental writers have published cases.

Of late, Gowers<sup>12</sup> has embodied his carefully made and valuable observations in a well written volume in which the history of the disease is illustrated by brief reference to the cases reported by Continental, English and American authorities, one hundred and seventy-six in number. Of these, all but eight were among children.

**Symptoms**—Duchenne details the symptoms in the following order:

1. In the beginning, feebleness of the lower limbs.
  2. Lateral balancing of the trunk and widening of the legs during walking.
  3. A peculiar curvature of the spine or saddle-back, both in walking and standing.
  4. Talipes equinus, with an over extension of the first phalanges of the toes.
  5. Apparent muscular hypertrophy.
  6. Stationary condition.
  7. Generalization and aggravation of the paralysis.
- These are the striking features of the disease, which is far from common,—and, so far I

<sup>1</sup> Quoted by Draper, Am. Psy. Journal, Feb., 1876.

<sup>2</sup> Op. cit.

<sup>3</sup> Nervous System of the Human Body, etc., 2d Ed., 1830, 3d Ed., 1836.

<sup>4</sup> Referred to in Schmidt's Jahrbuch, xxiv., p. 176 and by Gowers.

<sup>5</sup> Transaction of Medico Chirurgical Soc., xxxv., 1852.

<sup>6</sup> Archives Général de Med., January, 1868, and following numbers.

<sup>7</sup> Appendix to Aitkin's Practice of Medicine, 1868.

<sup>8</sup> Boston Medical and Surgical Journal, Nov., 1878.

<sup>9</sup> Philadelphia Medical Times, June and July, 1871.

<sup>10</sup> Photographic Review, Oct., 1871.

<sup>11</sup> New York Medical Journal, June, 1875.

<sup>12</sup> Pseudo-Hypertrophic Muscular Paralysis, a clinical lecture, London, 1879.

have seen less than a dozen cases. In illustration of the development of the disease, I may present the history of a well-marked case which I was permitted to examine by Dr. V. P. Gibney.

F. S. M., aged 13. Previous health excellent, her only illnesses being whooping-cough at the age of 9 months, and scarlatina one year ago, which was followed by some otitis. Her family history is good, so far as nervous disease is concerned. Her father died of phthisis, and her mother is alive and healthy. Her ancestors were long-lived people. She tells us of an injury received in 1870, a boy having thrown a brick at her, which struck her in the small of the back. No fever or pain preceded her present trouble. Her disease was of gradual development, and the hypertrophy followed the injury which has just been alluded to. At the end of six months she found it difficult to go up stairs, and her helplessness increased until the time of admission into the Hospital for Ruptured and Crippled, April 7, 1876. The following history was then taken: Complexion, light; hair, brown; eyes, hazel. She is small for her age, though well developed. She stands with abdomen prominent, chest and head thrown backwards; walks with an unsteady, waddling gait. Upper extremities, with exception of elbow-joints, which permit extension beyond an angle of  $180^{\circ}$ , normal. From the sixth dorsal to the sacrum there is a lordosis of three inches, the point of greatest incurvation being at the third lumbar vertebra. There is tenderness on deep pressure over the twelfth dorsal vertebra, while both trochanters stand out prominently, and the limbs are widely separated, and there seems to be no trouble about the hip-joints. There is marked diminution in power of the extensors of the legs, preventing her from holding the limb at a right angle to the body. There is no marked loss of power in the flexors. But there seems to be some loss of power in the anterior foot muscles; no comparative atrophy of limbs. The muscles of the back seem small and poorly nourished. The girl has difficulty in arising from, or assuming the sitting posture. The lordosis can be overcome by the voluntary act of stooping forward.

*Treatment.*—Spinal brace and electricity.

Through the kindness of Dr. Gibney, I was permitted to examine the patient, whom I found to be a rather well-nourished girl. I was immediately struck by her gait, which was characteristic of pseudo-hypertrophic paralysis. The feet were planted widely apart, and when propulsion was attempted, the whole pelvis was seemingly twisted, and the legs clumsily swung forward. The body swayed from side to side, the abdomen was prominent, and the shoulders drawn back, so that the extreme lordosis described so clearly by Duchenne was very beautifully shown. When stripped, this exaggerated curve was found to be very great. A plumb line held at the seventh cervical spine fell about four inches back of a line drawn across the upper edge of the sacrum. When my hand was placed upon her abdomen, and an attempt was made to force her to stand erect, the nates were immediately thrown backwards, and she would have pitched forward if not supported. When she attempted to walk, the pelvis seemed to be lifted on the side of the limb which was raised, and at the same time the corresponding side of the abdomen became quite flat. Her gait was waddling, and she progressed very slowly. There was some spinal tenderness, but no other disturbance of sensibility either in the sound or hypertrophied muscles. The latter were those of the back of the leg, which were much larger on both sides



than they should have been, and were quite hard and in marked contrast to the other muscles of the body, which were flabby and poorly nourished. The muscles of both thighs at the inner side seemed to be atrophied, as were all the muscles of the back; but the arms were of normal contour, and apparently unaffected. There was considerable loss of power in the lower extremities, the patient being unable without great effort to rise from her chair, and when she attempted to do so, she planted her feet widely apart and approximated her knees. The color of the skin was rather darker than it should be, and especially on the feet, legs, and hypertrophied calves, was there mottling and imperfect incubation. No difference in tactile sensibility could be noted. Measurements of different parts gave the following results:—

|  |            |
|--|------------|
| About shoulders . . . . .              | 29 inches. |
| About waist . . . . .                  | 24 "       |
| Middle of right thigh . . . . .        | 14 "       |
| Middle of left thigh . . . . .         | 13½ "      |
| Right thigh, just above knee . . . . . | 11 "       |
| Left thigh, just above knee . . . . .  | 12 "       |
| Right calf . . . . .                   | 12 "       |
| Left calf . . . . .                    | 12 "       |

A case reported to me by my friend Dr. G. H. Swazey is the following. This patient was also seen by Dr. J. Lewis Smith:—

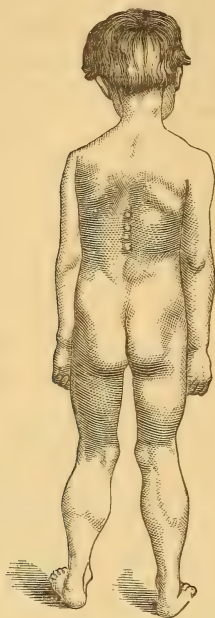
J. D., aged 2 years 8 months. Has always been a healthy boy until four weeks ago, when it was noticed that he seemed weak in his legs, especially in the morning, or after sitting awhile. Has not complained of any pain. When the child walks, it is in a peculiar wabbling sort of a way, with his legs wide apart, and his shoulders carried well back. He cannot stand well with his legs close together, but soon totters and falls. After he has walked awhile this peculiarity of gait is not so perceptible. The left leg measures around the calf eight and one-eighth inches, right leg around the calf eight inches. Just above the knee left leg measures nine and a quarter inches; right leg, same place, nine and one-eighth inches.

The weakness in the legs has been steadily increasing from the first. The grandmother of the child on the maternal side has epilepsy; and the grandmother on the father's side has what the mother calls weak spells, apparently of an epileptic character. An aunt and uncle on the father's side have epilepsy, and there is also a history of syphilis in the family. The mother has had miscarriages, apparently due to that cause. The father has had eruptions and other symptoms. March 28th commenced treatment with the faradic current to the muscles, which was continued three times a week for six weeks; the disease slowly progressing. At this time the patient left off coming, and has not since been seen.

Weakness of the lower extremities is one of the earliest symptoms, and is gradual in its appearance, and not preceded by fever, as is generally the case in infantile spinal paralysis. This impairment of power may begin imperceptibly, and first attract the attention of the parent by the inability of the child to walk at the usual time, or may appear subsequently, the child falling frequently or moving clumsily. In Poore's collection of 85 cases, it is shown that "3 never walked at all, 24 never walked well,

1 is reported as coming on gradually, 52 walked well at first, and in 5 cases no mention is made of the period of walking." "Of those who walked well, 2 began to walk at eighteen months, 3 at two years, 3 at two-and-a-half years, 4 at four years, 1 at five, and 5 are reported as walking late and badly."

Fig. 47.



(Gowers) Pseudo-Hypertrophic Paralysis.

Duchenne and Drake reported cases in which convulsions were the beginning of the disease. Pain in the calves of the legs or back is sometimes the first symptom, but is by no means one to expect as a rule. The appearance of the patient is most striking. The belly seems to be thrown out, the lumbar curve is increased, and the feet are widely separated. When the child attempts to walk, his movements are very much like those which we might expect to see in an individual laboring through a quagmire. There is a certain amount of waddling, the legs being separated, and the feet planted at some distance apart. In progression the body is inclined to the side on which the foot is planted, and there is some jerk made in the effort to carry the foot forward. The patient rises from the sitting posture with some difficulty, as there is great impairment of the extensor muscles of the spine. This weakness is the cause of the difficulty in keeping his balance. The next stage of the disease is the development of the hypertrophy. Very often this change is an early one, and may follow closely after the commencement of the impaired motor power. The calves are generally first enlarged, and this enlarge-

ment may begin with the difficulty in walking, or within a period anywhere from six months to several years after the beginning of the disease. This enlargement is not, however, always confined to the calves, but may affect the other muscles of the lower extremities, or even those of the upper. The glutei, gastrocnemii, deltoid, and many other muscles have been involved in cases reported by different observers. When the muscles are contracted, they stand out quite prominent, and in one of the cases reported by Barlow<sup>1</sup> the child's appearance resembled that of the Farnese Hercules. The child is unwieldy and awkward, and though there is at this stage some increase in strength of some of the members used in locomotion, the child does not seem to have very much motor power, for he can scarcely walk. The muscles not hypertrophied may undergo an atrophic change, greatly adding to the deformity. In regard to the talipes that may be produced, the extensors are agitated by spasmodic contractions, which become more aggravated as the attempt to walk is persisted in, so that, after a few steps, the child is quite likely to fall. Dr. Gowers has devoted much time to the discussion of the subject of muscular enfeeblement as a symptom.<sup>2</sup> He alludes to certain peculiarities of the patient's behaviour, which are striking and pathognomonic. One of these is the manner in which the patient arises from the floor. Owing to the weakness of the muscles of the back, the little patient always places his hands on his knees, "apparently to push the trunk up, to help the extension of the hip-joint." This, Gowers says, is met with in no other affection, and I am inclined to agree with him.

He first places his hand on the knee-joint, and when the knees are extended he works his way up, putting his hand upon his trunk until he effects extension of the hip.

"The reason why this action affords such help in extension of the knees, says Gowers, is obvious on a little consideration. In rising from the ground with the knees flexed, the weight of the trunk, resting on the hip-joint, is at the extremity (Fig. 48, W.) of a lever (the femur) of the third order, the fulcrum (F) being at the knee, and the power, the contraction of the quadriceps extensor, being applied (P) between the weight and the fulcrum,—i. e., in the position in which it acts to least advantage. But by placing the hands on the knees,—i. e., on the end of the femur,—a large part of the weight (the larger the more the patient bends forward) is transferred to the lever (at W) close to the fulcrum; the lever is, in so far, transformed into one of the second order, in which the weight is between

<sup>1</sup> Op. cit., p. 11.

<sup>2</sup> Even so far back as 1830 Sir Charles Bell \* recognized this as a striking symptom. "The paralytic debility of the muscles came on gradually: he was first sensible of it at a public school, about eight years ago. It began with a weakness in the thighs, which disabled him from rising; and it is now curious to observe *how he will twist and jerk his body to throw himself upright from his seat*. I use this expression, for it is a different motion from that of rising from the chair."

\* Op. Cit. Third Edition, p. 432, case clxxx.



the power and the fulcrum, and the power is economized in the greatest degree. Moreover, if the patient bend down, the centre of gravity may even be carried in front of the knees, and then, if the hands grasp the knees firmly, the weight of the body, instead of being the weight to be moved, becomes a force applied to the upper end of the femur, effecting the extension of the knee without the slightest action of the quadriceps ex-

Fig. 48.



tensor, as any one may ascertain by observing the mobility of the patella in this attitude." The skin may often be greatly discolored in patches just as it is in infantile paralysis, and Duchenne has called attention to this *mottling*, which is due to modified cutaneous circulation, and is seen especially during the later stages of the disease. It is more often confined to the lower extremities, and the patches which at first appear as bright red discolorations gradually become more dusky as they are exposed to the air. This mottling is increased by muscular action, and in certain regions was found by Benedikt to be connected with local sweating. The temperature of the hypertrophied muscles is higher by a degree or two than those that are atrophied; and in the earlier stages electric contractility is rarely affected, but in the later it is greatly diminished. Of course this depends upon the fatty substitution which the muscular tissue has undergone, for but a small amount of normal muscular fibre remains to be called into action by the electric stimulus. Putnam, of Boston<sup>1</sup> reports a case of pseudo-hypertrophic paralysis with involvement of the tongue, which was broad and thick, and the face was smaller than it should have been. These conditions existed in addition to hypertrophy of the legs and thighs, back and arms—it is rare, however, to find involvement of the face.

<sup>1</sup> Bost. Med. and Surg. Journal, Jan. 3, 1880.

Gowers<sup>1</sup> presents some cases of the disease in adults. The examples of lipomatous myo-atrophy, given by him, are seven in number. In all the disease began after twenty, and in several after forty or thereabouts—two being females. In two cases, those reported by Barth and Müller there were autopsies made,—evidences of lateral sclerosis were found, and degenerative changes in the ganglion cells of the anterior cornua were disclosed. The lower extremities were affected in all the cases, though in several the hypertrophy was found in the upper as well. In three cases there was mental derangement.

**Causes.**—Beyond the question of heredity it is impossible to go in our search for causes. One or two cases, however, are mentioned by foreign observers in which injury preceded the disease. Kesteven<sup>2</sup> reported one of these, and in this case the hypertrophy appeared at the fifteenth year.

Poore's table<sup>3</sup> includes the following examples of heredity:—

"In two cases a maternal uncle and aunt had this disease.

"In one case three maternal uncles and aunts had this disease.

"In one case one maternal uncle and one half-uncle had this disease.

"In one case three maternal half-brothers had this disease.

"In one case a maternal half-brother, three maternal uncles, and other members on the mother's side, had shown the symptoms of pseudo-hypertrophic paralysis.

"In thirty-seven instances, two or more belonged to the same family. It will be observed that it is only on the mother's side that this hereditary influence is transmitted; while the disease shows itself almost exclusively in the males; thus in a case reported by Duchenne, the mother, while she escaped, transmitted the disease to the children of her marriage. The same fact is stated in Foster's case.

"In one case a maternal grandfather was hemiplegic.

"In one case a paternal grandfather was insane.

"In one case a father was insane.

"In one case a father was intemperate.

"In one case two brothers died of granular meningitis.

"In one case a brother was an idiot.

"In fifteen cases of the eighty-five the family history was good.

"In thirty-three cases no mention of family history is made."

Like other spinal troubles it is found that several members of the same family may be afflicted.

Drs. Steele and Kingsley<sup>4</sup> of St. Louis have reported several cases of pseudo-hypertrophic paralysis. Dr. Steele's cases were two brothers, and Dr. Kingsley's two sisters, aged ten and thirteen years. I have seen two cases in the same family, both of whom were girls, one being ten years old, the other seventeen. The youngest girl presented the lumbar curve

<sup>1</sup> Op. Cit., p. 62.

<sup>2</sup> Journal of Mental Science, vol. xvi., April, 1871, p. 48.

<sup>3</sup> Loc. cit.

<sup>4</sup> Reported in "Alienist and Neurologist," Jan., 1880.

and arose from her chair with difficulty. Her thighs were firm, but smaller than they should be, but the calves and nates were hypertrophied and hard, and it was impossible to take up any considerable amount of tissue between the fingers. She arose with difficulty from her chair. The older sister was helpless and could neither walk or stand, and in her case the disease had begun about the third year. I have also been informed of a family in which five children are affected.

**Pathology and Morbid Anatomy.**—According to Barlow, the first examination of muscular tissue in pseudo-hypertrophic paralysis was made by Griesinger and Billroth in 1865. Griesinger excised a small portion of the left deltoid, which was hypertrophied and paralyzed, and microscopically examined the muscle, which resembled adipose tissue. He found the fasciculi in a perfect state, but surrounded by fat. Eulenburg<sup>1</sup> and Conheim<sup>2</sup> found the muscular fibres reduced to fully one-sixth their normal size, and in some localities there were masses which they supposed were the sheaths of empty sarcolemmæ.

Auerbach<sup>3</sup> found hypertrophy of the muscular fibres, and an increased development of nuclei, but no interstitial fat deposit; but this was in a patient who died during the early stages of the disease. Berger's<sup>4</sup> experience was identical in an early case. Charcot<sup>5</sup> examined a case (that seen by Berger), and found the psoas in a state of primary alteration. The primitive muscular bundles were separated by broad spaces of connective tissue containing cells of a spindle shape, and nuclei. Other muscles were likewise affected. The pectoral muscles, and those having a sacro-lumbar attachment, containing fewer nuclei, and the internuclear spaces were filled with wavy connective tissue. In muscles which had undergone still more advanced degeneration, there was some evidence of fatty deposit. In this case he witnessed three stages of degeneration. In the earliest there was atrophy of muscular bundles, indistinct longitudinal striæ, and sometimes transverse striæ. The sarcolemmæ were filled with a hyaline substance.

Duchenne<sup>6</sup> denies the existence of empty sarcolemmæ, and regards the enlargement due to an increase of connective tissue containing fat-cells. Dr. Gowers has made an autopsy which revealed a condition of affairs strikingly like that found by Charcot. The gastrocnemius muscle resembled a fatty tumor, "a yellow, greasy mass of fat, in which no trace of muscular redness could be perceived." The muscular fibres presented no granular degeneration, but ran through masses of fat-cells with more or less fibrous tissue intervening. In the "narrow fibres the transverse striæ were farther apart than in the wider fibres." Various observers have

<sup>1</sup> Archiv für Heilkunde, 1865.

<sup>2</sup> Verhandlung der Berliner Med. Ges. i., pp. 101-205.

<sup>3</sup> Virchow, Archiv., vol. iii. p. 224.

<sup>4</sup> Deutsche Archiv für Klin. Med., 1872, p. 303.

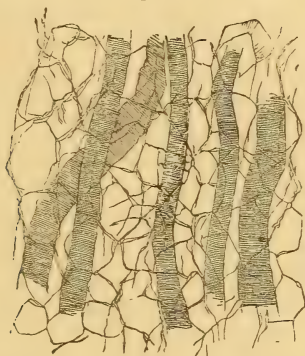
<sup>5</sup> Archives de Physiol., etc., 1872, p. 1.

<sup>6</sup> De l'électrisation localisée, Paris, 1872, 3d edition, p. 604.



examined the cord without finding any characteristic sign of trouble. The motor-cells have as a rule been enlarged. Gowers rather adopts the view that pseudo-hypertrophic paralysis is primarily of peripheral origin, and refers to the observations of Tschirjew, who found that the sensory nerve fibres end in the interstitial fibrous tissue, and that the posterior nerve-roots were those generally affected in this disease. He therefore traces some connection between these facts, especially as the fibrous tissue is the primary seat of the changes. He holds that there is an ascending degeneration.

Fig. 49.



Appearance of Muscular Tissue. (Charcot.)

Hitzig found an extraordinary increase in size of the arm of an adult, after injury near the shoulder joint, and the changed condition of the muscle was in every way like that of pseudo-hypertrophic paralysis.

In this case it was possible that there was an ascending neuritis, but it is also possible that the cerebro-spinal influence upon nutrition was suspended, while sympathetic system exerted an influence which gave rise to an increase in fat deposit. The effect of certain kinds of injury or irritation is witnessed in various pathological processes, which are characterized by the rapid formation of new tissue or phenomena of nutrition. The existence of hypertrophy and atrophy, at different stages of the same process, seems to me to be, in one instance, the commencing peripheral lesion, and in the other the result of a consecutive cerebral change.

**Diagnosis.**—Progressive muscular atrophy seems to be the only disease with which this condition may be mistaken. If the patient is seen at a time when the conditions of atrophy and hypertrophy coexist, it is not always easy to tell whether there is an increase of volume, or simply an atrophic condition of some muscles, while others are of normal size; but the other symptoms, alluded to, the exaggerated lumbar curve, and the waddling walk, should settle the question of diagnosis. Progressive muscular atrophy is also generally a disease which rarely appears at so early a period as does pseudo-hypertrophic paralysis. Increase of size

from determination of blood to a muscle, such as that reported by Maunder,<sup>1</sup> and sometimes fatty development, without paralytic symptoms, may deceive the incautious.

**Prognosis.**—The disease is slowly progressive, and death occurs generally from some other disease. Poore reports thirteen deaths. Phthisis, pleuro-pneumonia, uncomplicated pneumonia, and croup appear to have carried off most of these cases ; and it seems as if pulmonary disease bore some special relation to organic disease of the cord, particularly when trophic disorder accompanies such disease. In several of the spinal affections, especially when the anterior cornua are affected, there is generally the development of phthisis or other pulmonary maladies. The deaths that have been reported occurred rarely before the eighth year of the disease, and generally between the fourteenth and thirtieth.

**Treatment.**—Duchenne reports two cures by the faradic current. This seems to be the only remedial measure that promises anything very encouraging. In the previous edition of this book I advised the abolition of fatty food. This I believe was a mistake, for when we remember that the nourishment of nervous tissue is more perfect when we consume fats it will be patent that they are serviceable. In fact an enlarged experience teaches me that the case will not do so well when fat does not form a part of the dietary. *Massage* should be employed at least every day. The well-known fact that phosphorus produces fatty degeneration should contraindicate its use. Systematic exercise with wooden dumb bells, and calisthenics are to be indulged in, and the patient should be made to walk for a short time every day. As to mechanical support not much is to be said. Gowers recommends Sayre's jacket, which I think in a few cases is excellent. The children who suffer for want of support of the vertebral column when the muscles of the back are weakened may be greatly helped by this or some other form of bodily support. Arsenic and mercurials have been of service in the hands of some practitioners, among them Meryon.

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<sup>1</sup> Med. Times and Gazette, March 27, 1862.

## CHAPTER XI.

## DISEASES OF THE SPINAL CORD (CONTINUED.)

## POSTERIOR SPINAL SCLEROSIS.

**Synonyms.**—Progressive locomotor ataxia; Tabes dorsalis; Ataxie locomotrice progressive; Locomotor asynergia, etc.

When disease of the posterior columns of the cord exists we are furnished with a very interesting and striking train of symptoms, which are chiefly expressed by pronounced disturbance of the locomotory function, diminished reflex excitability and defects in co-ordination and sensibility. So delicate has the matter of diagnosis become that the coarse symptomatology of five or ten years ago is not essential to the recognition of the affection. It has been found that cases of so called "locomotor ataxia" may not be dependent upon disease of the posterior columns at all, but the symptoms occur as evidence of organic diseases of other parts, notably the pons. So, too, we meet cases of disease of the posterior columns without any of the pronounced locomotory troubles. Some of these patients are able to stand with closed eyes and do not walk with any peculiar stamp.

**Symptoms.**—Every pronounced case invariably presents three marked symptoms: 1. Peculiar pains usually seated in the lower extremities. 2. A simple atrophy of the optic disk. 3. An impairment of the reflex function, usually found in the tendon of the quadriceps, or shown in tardy action of the pupils. These symptoms are constant, but others are often found in conjunction.

Most authors have divided this disease into three stages: 1. That characterized by pains and commencing impairment of the tendon reflex. 2. That marked by the commencement and continuance of ataxic movements, etc. 3. The stage of decline in which the spinal lesion usually becomes extended, and various disturbances of nutrition are conspicuous, among them bed sores, general wasting of tissue, arthropathies, intercurrent phthisis, etc., etc. The first stage is usually the longest, and may last many years, or it may be almost inappreciable.

After exposure or prolonged dissipation, the individual may first notice the commencement of the disease in fulgurating pains which dart from the feet up the legs and thighs, and for the time he may suppose he has simply neuralgia or rheumatism. These pains are worse at night, and may be aggravated by damp or cold weather. They appear and disappear rapidly; and



Clarke<sup>1</sup> calls attention to their tendency to *move* suddenly from one place to another; remaining in one spot for some hours at a time, and then shifting to another. The pains are so prominent a symptom that they should never be disregarded. Some of the most advanced English clinicians go so far as to say that with the presence of fulgurating pains, absent tendon reflex and white atrophy of the optic nerves, they can infallibly diagnose locomotor ataxia even when all other familiar symptoms are wanting. The pains are explosive, inconstant and erratic, never following the course of any particular nerve, and there is none of the constant soreness or defined pain so peculiar to the various forms of true neuralgia of the lower extremities. They may shoot through the soles of the feet, the heels, the inner part of the legs, the knees, or even the thighs. After a time, which varies from a few weeks to several years, there may be a most disagreeable sensory change of a lesser grade, which is confined to the feet. When walking, the patient complains that "the ground feels as if it were covered with fur, or a padded cushion." Sometimes the sensation is likened to that produced by a stocking down at heel, or as if his shoe was filled with sand; or, again, as if he were walking in the air. There is no loss of muscular power, nor general loss of sensibility, in the preponderance of cases; but there only seems to be a perversion of tactile sensibility, and that only limited to the sense of contact. By far one of the most interesting of the general changes is the absence of the patellar tendon-reflex. Enough has already been said about the importance of this symptom, and it remains for me to add that in the greater number of cases it is absent, though I do not take the extreme view held by many authorities. In a number of instances I have found it exaggerated instead of diminished; but I am now inclined to think that where it is aggravated there is an extension of the disease to other parts of the cord. In the majority of cases of locomotor ataxia therefore no response follows the blow upon the ligamentum patellæ and no dorsal clonus can be evoked by bending the foot. Heat and cold are appreciated, but the shape or size of the cold or warm object cannot be perceived by the tactile sense alone. Painful impressions are appreciated, but this is all. Circulation becomes sluggish in the limbs, and subjective cold is felt in the lower extremities. If the individual is seated, and the hand of the examiner be held against the sole of the foot when the thigh is flexed, it will be found that he is generally quite able to extend the leg forcibly, but there may be sometimes a slight loss of power in subsequent stages when the anterior parts of the cord become affected. In the early stages of what may be called the *descending* form, there are various ocular troubles. Amblyopia, strabismus, or diplopia are among the more common, and it is not unusual to find some atrophy of the optic disk of either one or both eyes. In both forms of sclerosis of the cord, *ascending* as well as *descending* (these

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<sup>1</sup> St. George's Hospital Reports, 1866.

terms being applied with reference to the fact whether the disease begins at the upper part of the cord or *vice versa*), it is necessary for the patient to look at the objects which surround him in order that he may preserve his equilibrium. If he shuts his eyes, he is apt to topple over; and it is utterly impossible for him to walk in the dark without holding on to something for support. The patient very often finds that when he closes his eyes, as he is about to wash his face, he is quite apt to pitch forward against the wall. This test is an important one, and if he is able to stand with his heels and toes approximated and his eyes shut, it may be inferred that either his disease has not advanced to a serious extent, or that it is not locomotor ataxia at all. The early ocular trouble is strabismus, which is an inaugural symptom, and is very often accompanied by amblyopia; and if the strabismus be single, the amblyopia will be on the same side. The pupils are sluggish, and sometimes are entirely insensible to light. They are as a rule both contracted, though they may be unequal. Jackson, alluding to this state of the pupil, which he calls "Argyle Robertson's symptom," states that he believes it to be due to a loss of reflex activity, and but a link in the chain of disordered functions, which in the lower extremities is expressed by the absent tendon reflex. In eight cases reported by me, the tendon reflex was absent in four, and in two of these subjects there was neither impairment of vision nor any ocular troubles whatever; but in the other two there were both optic nerve atrophy and pupillary changes, one woman having pin-point pupils.<sup>1</sup> Various paralyses of cranial nerves may also follow, and ptosis is not an unusual symptom. Nothnagel<sup>2</sup> publishes the notes of a case where hyperæsthesia of the parts supplied by the fifth nerve was a prominent symptom. The lost power for localization is not uncommonly associated with this disease. With closed eyes, the individual is unable to place the tip of his finger on his nose, or upon any desired small point; and, when told to touch the point of a pin held by an observer, he will be unable to do so, his finger missing the mark. When awaking, he is often undecided as to the whereabouts of his legs, or sometimes feels for a moment that he has none, and needs the aid of vision to see that there are such members. The nerve-fibres in the posterior columns lose their facility for the conduction of sensory impressions; and it is sometimes several seconds before an impression made at the periphery is received at the sensorium, and appreciated by the individual. A symptom sometimes found in this disease, as well as in myelitis, is the sense of constriction which is referred to the waist. The bowels, in the early stages, are generally confined; and there is some loss of control over the bladder, and constant desire to empty that organ. Romberg calls attention to the fact that the stream seems to have no force, but falls to the ground on leaving the meatus. The individual is also troubled by erections during the early stages, and

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<sup>1</sup> Boston Med. and Surgical Journal, Dec. 19, 1878.

<sup>2</sup> Berlin Klin. Woch., xvii., 1865.

there is greatly increased sexual power. This, however, is diminished towards the end of the disease, and in males impotence follows.

Müller<sup>1</sup> has noticed certain peculiarities in regard to locomotor ataxia which have not been fully noticed hitherto. He speaks of the urethral and rectal neuralgias, which are connected with tenesmus, and may be mistaken for other trouble. He also calls attention to the severe coughing paroxysms that indicate affection of the pneumogastric, and he has also found that it is impossible, even by the use of pilocarpine, to induce sweating in the affected limbs.

Irritability of temper, occasional mental disturbance, and loss of memory are not rare evidences of intellectual failure, and occur at different stages. The electro-muscular irritability seems to be rather increased than diminished. The locomotory trouble appears quite early, and is one of the most distressing features of the disease. It begins by an awkwardness in progression, and the feet fly out and are planted with a kind of jerk, the heel touching the ground first. The individual totters, and is eventually unable to walk at all without support, and the gait cannot be mistaken by any one who has once witnessed it. The sense of appreciation of weight also seems to suffer to a decided degree. Jaccoud<sup>2</sup> found that this is lost to a great extent, and that there is a variation in the power to perceive weights on the two sides of the body. In one case mentioned by him, a pressure equal to 3000 grammes was perceived on the right side, and 2800 on the left. The pains before spoken of generally disappear as the disease becomes confirmed, though they may last throughout. Fibrillary contractions are occasionally seen; and, speaking of this, I have often witnessed a curious phenomenon which follows the use of faradism. I have noticed that when a muscle of one leg was agitated by clonic contractions, sometimes the same muscle in the other leg would be contracted synchronously with that under electric stimulation. The patient is generally timid, and easily disconcerted by any sudden noise or unexpected excitement; and when crossing the street, the desire to avoid being run over on the approach of a wagon will produce such demoralization as to prevent him from taking another step, and he sometimes falls to the ground. There is rarely trembling, unless the disease has involved the upper part of the cord, when this symptom, as well as the inability to appreciate topographical points, will be marked. The patient is generally worried, anxious-looking, and woe-begone, and is full of complaints. The disease may last for from five to twenty years, and the patient is carried off by tuberculosis or some intercurrent pulmonary affection. Atrophy of all the muscles of the extremities generally takes place towards the end of the disease, and bed-sores and arthritic troubles are annoying and painful forerunners of death.

Charcot has called attention to certain cutaneous eruptions which not infrequently are found with posterior spinal sclerosis, and which are

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<sup>1</sup> Abstract in "Brain," vol. 3, No. 4.

<sup>2</sup> Op. cit., p. 341.



usually of a papulous and pustular character. He mentions the case of one person, who, while under treatment at La Salpêtrière, presented large patches of urticaria, the appearance of which was coincident with the attacks of pain. Other writers have called attention to the existence of herpes in connection with the pain, and I myself have found patches of this eruption in connection with the early severe pains especially on the inner surface and back of the thighs.

The eruptions generally mark out the course of the nerve which is the seat of pain; Hutchinson, however, considers that this arrangement of the eruption is usually misinterpreted, and that, instead of the eruption following the direction of a nerve-trunk and its branches, the corymbiform distribution of the skin-disease in reality corresponds with the course of the small vessels.

Occasional but exceedingly interesting features of the disease are the joint troubles and certain trophic alterations in bone-tissue leading to decided brittleness and liability to fracture. Charcot was first to call attention to these symptoms, and Buzzard, Weir Mitchell and others have written extensively about them since. Arthropathic changes may begin at any period of the disease, but are more common during the last stage. The joints of the lower extremities seems to be more frequently the seat of swelling than other parts, and this is true also, as Arnozan<sup>1</sup> points out, regarding the brittleness of bones, those of the trunk rarely undergoing change.

The knees or ankle joints may be the seat of a cold, puffy, soft swelling of gradual growth, and nearly always attended by no increase in temperature, pain or evidence of inflammatory action. After a time it is possible to detect a much greater degree of mobility, which is due to loss of substance, and it is an easy matter to twist the limb or dislocate the bone. At an early stage of the affection the patient may find it impossible to stand, because of the "turning" of the ankles. This is the case in one patient I have under observation; but I regard a double symmetrical arthropathy a rare condition. It is possible to hyper-extend a limb, so that, for instance, a distinct fold of skin may be perceived upon the anterior aspect of the knee below the patella when the leg is carried forward, the patient's extremity presenting the appearance of that of a child's doll. The foot may be everted to a great degree, or the thigh dislocated with great facility. The muscles about the arthropathy are often atrophied and feeble, and do not keep the limb in place. The articular surfaces may be felt, and will be found to be greatly reduced in size.

Charcot and Raymond,<sup>2</sup> in alluding to the disappearance of the heads of the long bones, relate the case of a woman, aged 52, who had been ill for many years. The autopsy revealed atrophy of the different processes of the humerus, femur, tibia, and scapula, with muscular degeneration of a fibrous character. In another case there was hip-joint affection, and

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<sup>1</sup> Des Lésions Trophiques, p. 86, 1880.

<sup>2</sup> Gaz. Médicale de Paris, Feb. 19, 1876.

great brittleness of the bones, which broke when subjected to inconsiderable force, and afterwards united quite readily. During life the evidences of such arthropathies are sometimes numerous. They may be illustrated by the following case of Bourcere.<sup>1</sup>

The patient was a woman who entered La Charité April 8, 1875; she was middle-aged, and presented many of the symptoms of locomotor ataxia. These began about ten months before. The left leg seemed to be more affected than the right. Three days after admission the left thigh and buttock began to swell rapidly, and in a few hours the swelling, which was not œdematous in the strictest sense of the word, but hard and not painful on pressure, reached its maximum. It extended as far down as the knee, where it stopped abruptly. There was no fluctuation, nor any evidence of pus. The swollen part was almost double the size of the other limb, while the leg was shortened, and the foot was to some degree rotated outwards. There was also some swelling and hardness unattended by tenderness in the left iliac fossa. The swelling disappeared almost entirely in a week, when vaginal examination was made, and a hard, smooth tumor was discovered, which apparently sprung from the pelvic bones of the left side. Pus was soon afterwards detected in the psoas sheath above and below Poupart's ligament. She became prostrated, and died on the 6th of May. After death decided osseous changes, to be hereafter described, were observed.

It has been found that in many subjects the bones undergo a chemical change which renders them liable to fracture. This fracture is spontaneous and may be caused by some such simple movement as crossing the legs suddenly. The accident may be preceded for some days by an increase in the violence of the fulgurating pains and perhaps by some redness and swelling at a point. It is not rare to find several fractures occurring after each other but there seems to be rapid repair.

Locomotor ataxia may be associated with progressive muscular atrophy, or may sometimes terminate in general paresis of the insane. Westphal and Obersteiner, have written much upon the relation of the two diseases and their possible coexistence.

Obersteiner,<sup>2</sup> in an excellent paper upon locomotor ataxia and mental diseases, considers that mental symptoms are found in the greater proportion of cases of this disease, and calls attention to the fact that these expressions of psychical trouble may be very slight; still, an acute observer will know that there is a departure from the normal intellectual condition. The patient's character is changed markedly. I have been often astonished at the apathy of an individual, or, on the other hand, at his irritability of temper, the violence of his anger, and his petulance, which are more than transitory evidences; and they are as important symptoms, I think, as neuralgic pains, difficulty of co-ordination, etc. These changes were all well displayed in a patient of my own; in health a most amiable, high-minded person; in disease a morbid, bad-

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<sup>1</sup> Progrès Méd., Oct. 9, 1875.

<sup>2</sup> Wiener Med. Woch., No. 29, 1875.

tempered, whining wreck. He had been noted for his gallantry on the field during the war; but after his disease had become established, his character seemed to undergo a complete transformation. He wrangled with every one, became irritable over petty things, and made himself generally disagreeable.

Obersteiner and Simon<sup>1</sup> both agree that these patients should be examined most carefully, and that the prognosis depends much upon the facts relative to mental alteration. The latter says: "It is not enough that the patient keeps himself quiet, and answers the questions relative to his age, how he feels, etc., and does not show marked delusions;" these are not enough to assure us that his intellect is intact.

In regard to the grave secondary mental changes, Tigges considers general paralysis to be a complication, while Obersteiner is convinced that the symptoms of this latter disease indicate a progression of the sclerosis upwards. He considers the lesions to be identical, and that it is only the seat of the change which has anything to do with the symptom expressed. He has also found, in general paralytics who have died, a sclerosis of the cord.

M. Rey has observed nine cases of insanity associated with locomotor ataxia. In three of these the spinal sclerosis preceded the cerebral trouble, and in one the induration had extended from the posterior to the lateral columns. He found that the diagnostic difference between locomotor ataxia combined with cerebral induration, and simply descending general paralysis of the insane, was the walk. In the former the patient could not stand with his eyes shut, and in the latter there was no difficulty of the kind. We may also take for granted that the walk of the ataxic is an early symptom, and that of the general paralytic a late one. Both are examples of defective coordination, and I think the latter is unwisely called *paralytic*.

The difficulty of turning around is marked in ataxia, but it is not a prominent symptom in general paralysis. The individual walks steadily across the floor when told to do so, but when he has to retrace his steps, he spreads his feet, and if the loss of co-ordinating power be at all great, he falls if he has no support.

A case lately came under my charge where the sclerosis of the cord was ascending, and in an incredibly short time the cerebral symptoms which indicate the general paralysis of the insane were evident.

M. F., aged 29; United States. On admission to the Epileptic and Paralytic Hospital, March 6, 1876, I was immediately struck by the woman's walk, which was ataxic in the extreme; and on questioning her and her husband we ascertained that about two years ago she had neuralgic pains in the legs and feet; her walking became defective, and has continued so. Her mind was clear up to a short time. Her pupils are now unequally dilated, the left being the largest; her lips tremble distinctly.

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<sup>1</sup>Archiv. für Psychiatrie, i. and ii., 1875.



Her tongue, when protruded, also quivers; when told to keep it quiet, the motion is greatly exaggerated. There is some ptosis of the left eye. When told to close her eyes, she is unable to co-ordinate delicate muscular movements. She cannot find the tip of her nose with her forefinger by more than an inch. When her eyes are open, she cannot touch small points, such as the markings upon my watch-dial. When she stands with her eyes closed, she topples over almost instantly. When she walks, her toes are thrown out, and she comes down upon her heels. Her feet are planted far apart when she attempts to stand. When walking across the room, she reels, and has difficulty in turning around. When attempting to answer questions, she talks slowly, each word being uttered with some effort, the words containing the letter "f" and "p" are *explosive*, and the lips seem to have a great deal of work to form them. The consonants are slurred over; for instance, the word "man" is pronounced "mah;" the "l's" are dropped, as are many other letters. Her writing is very scratchy and irregular, although her husband says she formerly wrote an excellent hand. Mentally she is silly, and laughs immoderately at wrong times and without cause. She has no idea of time, but seems to know what she is saying. She has had several delusions, one of which was that she had been home the day before.

May 12th, two months after admission.—Her walk is much worse; no urinary or other difficulty. There is some festination; pupils still uneven. The difficulty in speech has markedly increased. Her tottering walk is striking. We at first thought she had syphilis, but this is not so. Being unmanageable and restless, she was transferred. Here, undoubtedly, was an ascending condition, beginning with the pains and gait of locomotor ataxia, and ending with several early symptoms of general paralysis.

Charcot has described a peculiar train of symptoms accompanying the pains of the earlier stages. These are the *crises gastriques*, which are expressed by pains which begin in the groins, and run up the abdomen on either side, finally becoming fixed at the epigastrium. They are violent, and occur during the exacerbations of lancinating pain in the lower extremities. During the time they last, there is violent palpitation, vertigo, and vomiting, the latter symptom occurring without relation to the condition of the stomach. If there be no food to be expelled from that organ, there may be a quantity of frothy and bloody liquid ejected. These *crises* last two or three days, and disappear quite suddenly. Buzzard has found that there is some connection between them and the arthropathies, and of nine cases with joint troubles, six presented the crises as a symptom. Some observers have noticed the appearance of ptosis during their existence, which gradually disappears. Stewart<sup>1</sup> has seen several cases in which these symptoms varied, and instead of there being pain which started from the groin, there was deep-seated pain in the dorsal and lumbar regions.

Raynaud has called attention to a species of renal neuralgia which is not at all an uncommon complication. One of his cases, which was mistaken for renal colic, presented lumbar pain, vesical tenesmus, retraction

<sup>1</sup> Med. Times and Gazette, Oct. 7, 1867.

of the testicle and other suggestive symptoms like those described by Müller. There was temporary cessation after a few days, but a second and third attack followed. Charcot and other French writers have alluded to various additional visceral disorders, as found with this as well as other organic spinal diseases, and the functions of the kidney are sometimes greatly disturbed. I do not think that sufficient attention has been paid to forms of hysteria which resemble locomotor ataxia. These, I believe, are the cases which are cured. Isnard<sup>1</sup> has extensively considered the functional form; and Webb and Mitchell, of Philadelphia, have reported very interesting cases of genuine hysteria which counterfeited the organic disease quite closely.

Diphtheria is sometimes followed by a nervous condition that is apt to be mistaken for true locomotor ataxia. Séguin calls attention to the fact that the ocular trouble consists in paralysis of the ciliary muscle and consequent dilated pupils, with loss of accommodation instead of the organic ocular change so marked in true spinal sclerosis posterior. This condition, too, is of short duration.

**Causes.**—Dissipation has much to do with the development of this terrible disease, while onanism and venereal excesses, especially play an important part; so that we may expect to find it among men about town, hard drinkers, and other people of bad habits. Injury, exposure to rain and cold; syphilis, and protracted mental excitement, favor its origin. These are rare cases, and I have seen one in which the disease suddenly appeared after injury, running a peculiarly rapid course. At the Hospital for Epileptics and Paralytics there is such a case in the person of a German workman who broke his femur, the fracture being simple. He was carried to the hospital and his injuries were treated in the usual way. After four or five weeks he began to have the fulgurating pains, and within four months there have appeared all of the pronounced symptoms of a grave case. He can hardly stand, and cannot walk without clinging to the sides of his bed. He has complete loss of the "tendon reflex," commencing optic atrophy, immobile pupils, difficult deglutition, etc. Petit,<sup>2</sup> in referring to the traumatic origin of the disease, does not allude to the rapid form, but contents himself chiefly with considering the influence of injury upon the established affection. He considers that falls upon the back, nates, or direct jarring of the cord transmitted by a fall upon the feet, are favorable to the development of the disease. Some sudden exposure, such as a fall into the water, or a night in the rain, may be the exciting cause, and several of my cases had such a beginning. Rosenthal<sup>3</sup> reported sixty-five cases, forty-six of which were males and nineteen females; and of this number thirty-one were traced to libidinous excesses, seven to exhaustion, and twenty-seven to cold and exposure. The youngest of these

<sup>1</sup> L'Union Médicale, 131, 134, 135, 137, 141, 142, 1862. Abst. in Lancet, Sept. 30, 1875.

<sup>2</sup> Revue Mensuelle, No. 3, 1879.

<sup>3</sup> Wien, Med. Woch., 1869, No. 251.

patients was nineteen, and the oldest sixty-eight. The ages at which the disease appears is rarely before the thirtieth, and never after the sixtieth year. Heredity seems to have much to do with its development, for instance, Friedreich<sup>1</sup> reports six cases which occurred in two families; and two of these patients were males, and four were females. The heads of the families were drunkards. Before the Clinical Society of London, Gowers<sup>2</sup> presented the histories of five cases of locomotor ataxia in the same family. The mother had had chorea in early life, but the father himself was healthy, though some of his brothers had been insane. There were nine children in the family. "1. A son, aged 39, with well-marked ataxy, which commenced at nineteen. He is just able to walk with crutches. There is inco-ordination of the arms and affection of articulation. Sensation to touch is normal, that to pain is in the legs increased. The knee-jerk is lost. 2. A girl who died of fever at ten years old. 3. A son, aged thirty-five, healthy. 4. A son, aged thirty-three, healthy. 5. A girl, aged twenty-nine, in whom the affection commenced at eighteen. She can now scarcely stand; there is weakness in the legs as well as ataxy, and also inco-ordination of the arms. Speech is affected, sensation is normal, the leg-jerk is lost. 6. A son, aged twenty-six, perfectly well. 7. A son, aged twenty-three, considerably affected,—the disease showed itself at nineteen. \* \* \* 8. A son, aged twenty-two, reported to be well, but found on examination to be distinctly affected. \* \* 9. A son, aged nineteen, affected in rather a greater degree than the last." These two cases showed all the early symptoms—inability to stand with eyes closed, absent tendon reflex, and confluent articulation. Friedreich and Dr. A. Carpenter have also presented cases—the latter, two cases in the same family; but it is questionable whether Friedreich's cases were true locomotor ataxia. Syphilis, as I have said, is sometimes at the root of locomotor ataxia, and perhaps is the most fortunate cause to discover, as it greatly alters the prognosis of the disease. It must be understood that the lesion is purely syphilitic; and the symptoms result simply from the presence of a gummy infiltration or tumor in the posterior columns, and not from any induced sclerosis. Erb is disposed to lay great stress upon the frequency of the association of syphilis and the disease under consideration.

**Morbid Anatomy and Pathology.**—The cord of the ataxic, when cut into, will present an appearance which is distinctive. The posterior columns will be found to be more gray and dark than they should be, and there may be hard deposits on either side of the posterior fissure. Beneath the microscope the peculiar thickening of the connective tissue will be found to have taken place at the expense of the nervous elements. Lockhart Clarke thus tersely describes the changes that take place:—"The morbid anatomy of locomotor ataxia consists chiefly of a certain gray degeneration and disintegration of the posterior columns of the spinal cord, of the posterior roots of the spinal nerves, of the posterior gray sub-

<sup>1</sup> Virchow's Archiv., xxvi., pp 391, 433. <sup>2</sup> London Lancet, Oct. 16, 1880, p. 618.



stance or cornua, and sometimes of the cerebral nerves. A variable number, and frequently in the latter stages of the disease nearly all the fibres of the posterior column and posterior roots, fall into a state of granular degeneration and ultimately disappear. Usually the posterior columns retain their normal size and shape in consequence of hypertrophy of connective tissue which replaces the lost fibres.

"In this tissue, at wide but variable intervals, lie imbedded the remaining nerve-fibres, with the *debris* of their neighbors in different stages of disintegration. In some places they are severed into small portions, or into rolls or lobular masses formed out of the medullary sheaths of white substance, which has been stripped from their axis cylinders. In other places they have fallen into smaller fragments and granules, which are either aggregated in the line of the original fibres or scattered at irregular distances. Corpora amylacea are usually abundant, and oil-globules of different sizes are frequently interspersed among them and collected into groups of variable shape and size around the blood vessels of the part. I am inclined to believe from my own investigations that in the course of the disease the posterior cornua of gray substance are more or less affected, and it appears to me to be a question whether they are not the first parts, or at least among the first parts that are morbidly changed. I have also shown that in some cases the deeper central parts of the gray substance are more or less injured by areas of disintegration. These latter lesions, however, are not essential to the production of locomotor ataxia, the peculiar symptoms of which depend solely on lesions of the posterior columns of the posterior nerve-roots, and probably of the posterior cornua. The cases in which they occur may be considered as mixed cases, partaking of the nature of locomotor ataxia and common spinal paralysis." Charcot and Pierret do not consider sclerosis of the fillets or columns of Goll to be the essential lesion of the disease under consideration. They rather hold that the degenerative process begins in the lateral parts of the posterior columns. It has been shown that the nerve-roots themselves need not necessarily be affected, although the cornua may be degenerated most completely.

Numerous interesting experiments have been made by Schiff,<sup>1</sup> Ludwig,<sup>2</sup> and others, some quite recently by Ott,<sup>3</sup> and G. B. W. Field,<sup>4</sup> in this country, that are likely to change our views materially, not only with regard to the pathology of this disease, but of many others. These authors, with the exception of the first-mentioned, hold that the lateral columns of the cord are the regions in which the conductors for voluntary impulses, inhibitory nerves, sudorific nerves, vasomotor impulses and sensations of pain are situated, while the posterior columns "conduct tactile impressions and co-ordination impulses." The gray matter, according to the carefully-made expe-

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<sup>1</sup> Lehrbuch der Physiologie des nervensystems, 1859.

<sup>2</sup> Ludwig's Arbeiten.

<sup>3</sup> American Med. Journal, Oct., 1879.

<sup>4</sup> Journal of Mental and Nervous Disease, April, 1881.

riments of Field, has no office in the conduction of any of these impressions. It would appear, then, that so far as definite co-ordination and impairment in the reception of tactile impressions goes that the posterior columns are concerned; but that the disease must involve the lateral bands of this region, and involve either commissurally or directly the lateral columns themselves, to give rise to the phenomena of pain that belong to locomotor ataxia. This agrees perfectly with the statement of Erb,<sup>1</sup> that "the typical form of tabes does not depend exclusively upon disease of the posterior columns of the spinal cord, but that other parts in the vicinity of the posterior columns must also be involved in the disease." If the columns of Goll are involved it will be later. The sclerosed parts of the cord in this disease are more commonly the lumbar and lower dorsal, although the cervical portion may be invaded as well. The case mentioned by Nothnagel presented sclerosis of the entire posterior columns.

The bones undergo remarkable changes before referred to, and after death the result of such arthropathic alterations may be seen in atrophy, exfoliation, shortening, and destruction of their articular surfaces. The appearance of old fracture is admirably shown in Fig. 50, which is taken from Charcot. A peculiar osseous change has been noted by

Fig. 50.



Appearance of Trophic Bone Changes in Locomotor Ataxia. (Charcot.)

Luys and others, and this consists in wasting of the alveolar processes so that the teeth lose their support and drop out.

The interest connected with the various phases of altered nutrition of bony tissue as a consequence of spinal disease, depend, to a great extent, upon the discovery of <sup>2</sup>Rauber and Talamon,<sup>3</sup> the first of whom discovered

<sup>1</sup> Article in Ziemssen's Cyclop., vol. xiii., p. 602.

<sup>2</sup> Centralblatt No. 20, p. 305, 1874.

<sup>3</sup> Revue Mensuelle, 1878, vol. ii.

corpuscular termination of nerves in synovial membranes and ligaments. What the exact nature of this connection is remains to be studied. Talamon reports a case of arthropathy in which there was no disease of the large cells in the anterior columns, and the researches of Charcot are equally unsatisfactory in pointing to the trouble as a result of the same processes which enter to so great a degree in such other diseases as infantile paralysis and the like. The conclusions of <sup>1</sup>Buzzard seem to throw light upon the subject, however. This writer, who, as has been stated, found that the *crises gastrique* were most frequent in patients who presented arthropathies, and that decided lesions of the radicular fibres of the pneumogastric probably existed, concluded that in the neighborhood there was another bulb or centre, which was likewise affected, and as a result the osseous changes occurred. <sup>2</sup>Arnozan is not disposed to accept Buzzard's view in their entirety, and is rather inclined to look for the lesion in the sensory region of the spine, and he is led to this opinion by the association of arthropathies, with an increase in the symptomatic pains in the extremities.

If Buzzard's autopsical results bear out the connection between disease of the nucleus of the pneumogastric, and the existence of crises and of arthropathies, it may raise the question of trophic changes as a result of general nutritive disorder. This seems plausible when we realize the fact that chemical alteration in the bones of ataxics has been found by <sup>3</sup>Regnard, who discovered that the phosphates had diminished in proportion, as the fatty matter had increased.

The fractures of the bones of ataxics are characterized by the rapidity with which union takes place, the exudation of callus being remarkably rapid, as was shown in Richet's example, who died a few weeks after a spontaneous fracture.

The cranial nerves are frequently affected, their course being interrupted by patches of degeneration. The induration attacks the periphery first, and extends to the centre, and the changes begin at the point of origin of the nerve and progress towards its distal end. The optic disk is nearly always found to be atrophied and blanched, but there seems to be no change in the size of the retinal vessels. There are often evidences of injection of the investing membranes of the cord or actual meningitis, and six cases which were reported by Friedreich presented opacity, and thickening of the pia mater, which was adherent to the cord; I doubt if there are many examples in which some form of meningitis has not existed at some time or other. Charcot<sup>4</sup> alludes to the gray degeneration of the optic nerves as an evidence of the amaurosis that is so prominent a symptom, and he calls the pathological condition "*névrite parenchymateuse*." Stilling has recently discovered a spinal root of the

<sup>1</sup> London Lancet, Feb. 7, 1880.

<sup>2</sup> Op. cit p 94.

<sup>3</sup> Gazette Médicale de Paris, Feb. 7, 1880.

<sup>4</sup> *Léçons sur le Syst. nerveux*, 2ème série, 1 fascic.



optic nerve which passes from the external corpus geniculatum, follows a deep course in the crus and is lost sight of in the medulla, and this suggests an explanation of the causation of the optic nerve atrophy even when there is no cerebral disease.

Much of the interest belonging to this disease is connected with the phenomena of inco-ordination, and a lesion that may affect the integrity of the organs intended for the transmission and reception of visual, auditory, or tactile impressions will result in a loss of equilibrating power. According to Ferrier, the apparatus provided for the maintenance of equilibrium consists of : 1, a system of afferent nerves ; 2, a co-ordinating centre ; 3, efferent tracks in connection with the muscular apparatus concerned in the action. Of course lesions of one or all of these parts must result in a loss of balancing power. Perhaps the most important factor in the preservation of equilibrium is tactile sensibility. The frog, deprived of his skin, loses the power of co-ordination, for the co-ordinating centre is deprived of the exciting organ from which impressions are transmitted. So, too, may this loss follow sudden destruction of one of the peripheral organs of special sense. As has been shown by Volkmann, the exposed ends of the nerves are not sufficient to transmit the sensory impression, but it is necessary that their cutaneous terminations shall exist. When the tactile sensation in the ataxic is blunted, or the impressions are interrupted in their upward course, as has been held by Schiff, we have a loss of co-ordinating power which is a striking feature of locomotor ataxia. It is not necessary for consciousness to enter into equilibration and co-ordination, for, as we well know, many acts are purely spinal in character, and become automatic to some degree ; and walking is notably one of these acquired automatic movements. Acephalous monsters have performed a number of acts which were strongly reflex ; and animals from whom the brains have been removed are able to co-ordinate to a certain degree after the first shock of the operation has passed by. In the disease under consideration consciousness enters to a decided extent when the harmony of the co-ordinating centres is lost. This consciousness is exhibited in vertigo, and is exerted in the ineffectual effort to regulate the actions of the limbs, the brain endeavoring to supply the lost automatic sense. Broadbent<sup>1</sup> considers that there are two co-ordinating centres ; one in the cerebellum, and the other, as I have stated, in the cord. Vision holds the same relation to the cerebellar co-ordinating power that tactile sensibility does to the cord centre. For instance, a tight-rope walker would fall were it not for the aid of vision, although the tactile sensibility becomes so perfectly educated that it may take the place of the eyes in enabling the performer to regulate his actions when he is blindfolded. The tactile sense is of a lower grade, and when this fails the individual, as is the case with the ataxic, requires more than ever the aid of vision. In the normal condition he may close his eyes, and still be able to walk in the dark with some ease ; but if the tactile sensibility be affected, as it is

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<sup>1</sup> Brit. Med. Journal, April, 1875.

in the disease under consideration, and if the aid of his vision be denied him, he is utterly helpless to regulate his muscular movements. In the daylight he still has the power of helping himself, for vision comes to his assistance. In health this delicacy of co-ordination may be trained to a marvellous degree. I have repeatedly witnessed the feats performed by a French juggler, which illustrated the nicety of appreciation of weight it is possible to arrive at by practice. He would throw into the air a heavy cannon ball and a pellet of paper, alternately catching them and tossing them up again, and the muscular movements were regular and harmonious, and indicated no effort whatever. In locomotor ataxia this power of appreciation is sometimes lost to a marked degree. To some ataxic individuals a four-pound weight seems no heavier than one of two pounds would if the patient were in normal condition, and if his muscular movements were properly co-ordinated.

One of the most interesting features of the disease is the question of absent tendon reflex. I have already expressed my doubts in regard to the universality of this symptom, but when the tendon-reflex is absent it indicates beyond all doubt a lesion of the cord above the third or fourth lumbar nerves as Prévost has demonstrated. Some authors believe the "tendon-reflex" to be purely a local phenomenon and among them, my friend, Dr. Augustus Waller,<sup>1</sup> of London, has advanced the idea that there is no such thing as a true spinal tendon reflex, basing his conclusion upon the fact that the appearance of the clonic spasm occurs too soon after the application of the stimulus. This he demonstrated by the myograph. He, therefore, considers that the phenomenon is due to a changed condition of the muscular contractility dependent upon some alteration in local innervation. Dr. Buzzard, on the contrary, in a series of elaborate papers, takes the opposite view, and says that it is a spinal reflex in every way, and that the shortness of interval between the application of the stimulus and the appearance of the contraction which is apparently inconsistent with phy-

Fig. 51.



The Course of Posterior Nerve-Roots.  
(Clarke.)

<sup>1</sup> "Brain" Part x. 1880.

siological mensuration of time, is quite possible when the sensibility of the nervous arc is exalted or in a favorable condition. He, therefore, can not take the physiological standard of time as the pathological. Prévost has in animals made pressure upon the aorta, and as a consequence the tendon reflex was abolished and did not return until the pressure was remitted.

The arrangement of the sensory fibers of the posterior columns is such that a lesion of either the white or the gray matter must interfere with the conductivity of sensory impressions. Lockhart Clarke's histological researches have thrown much light upon the subject. According to him, the posterior root-fibers enter the cord in three directions, some passing in at right angles to the longitudinal fibers of the posterior column, then passing across the same as well as the gray substance, then bending and continuing longitudinally downward, next passing into the gray matter of the anterior cornua, and finally terminating in fasciculi which intermingle with the fibers of the anterior roots, or extend into the anterior columns. Other fibers (those of the second class) run across the posterior columns, or cross to the other side of the cord in the posterior commissure, or extend deeply into the posterior columns of the same side; and others pass forward into the gray matter of the anterior cornua. The third kind of posterior spinal roots enter obliquely; and certain fibers pass upwards and downwards, and become associated with fibers above and below them. The remaining fibers take an oblique course, and run upwards and downwards, the greater number taking the former direction and passing finally into the gray matter. It will be seen that a lesion affecting the posterior columns of the cord will destroy the communication of the nerve-roots with the gray matter, or press upon the sensory fibres, causing peripheral pain. The communication with the parts above is destroyed, and should the sclerosis involve the anterior gray matter there may be paralysis and atrophy. A favorite theory, accepted by many writers, is that which considers that there are numerous centres of co-ordination in the cord, which are connected by longitudinal fibres, and that when these fibers are destroyed there results a species of inco-ordination. Dieulafoy<sup>1</sup> divided the posterior fasciculi at different heights, but without producing any marked defects in co-ordination, a result which seems to disprove this idea.

Onimus<sup>2</sup> explains the rigidity and awkwardness of the movements in locomotor ataxia by the theory that the stiffness of the muscles is perceived by the individual, and to overcome this he expends a greater amount of force than is needed for the particular act. The initial stiffness comes from the irritation of the anterior and lateral columns by the *mechanical presence of the deposit in the posterior columns*.

**Diagnosis.**—It is important to distinguish locomotor ataxia from *chronic myelitis*, *progressive muscular atrophy*, *chorea*, *cerebellar disease*, and *hysterical paraplegia*. The former occasionally resembles ataxia, but with ordinary care no mistakes need be made. The paralysis of

<sup>1</sup> Thèse de Concours, 1875.

<sup>2</sup> Gazette des Hôpitaux, July, 1878.



transverse myelitis is very marked, and the implication of the bladder and sphincter ani causes the patient to void his urine and feces involuntarily, which is not the case in locomotor ataxia. The strong ammoniacal odor of decomposed urine is itself almost a sufficient diagnostic mark. There is an absence of power in the legs, and none of the pain which characterizes sclerosis of the posterior columns. Ocular trouble and inco-ordination are likewise absent. If the gait of the two diseases be compared, it will be found that in the former the legs will be thrown out with some degree of violence, and the heel will come down forcibly. In the paraplegia of myelitis, the legs will be drawn after each other, the inner edge of the sole scraping the ground; and there is often a shrug of the body required to bring the feet forwards. The walk of the hemiplegic is also different, as one leg is swung forwards, the toe describing an arc, or else the foot is advanced in a straight line, the sole hardly clearing the floor. Myelitis in its early stages sometimes resembles posterior spinal sclerosis. The pain in the back, however, is characteristic, and the ulterior paralysis and bladder trouble are sufficient in themselves to clear up the diagnosis, though the constricting band about the waist may excite our suspicion. Cerebellar disease has been spoken of by Radcliffe<sup>1</sup> as a condition that may sometimes be mistaken for locomotor ataxia. The movements are somewhat different, however, for the patient rolls and sways to a greater degree, and does not present the peculiar jerking gait of the ataxic. Local pain is another symptom peculiar to the cerebellar condition, and vomiting is also suggestive of this affection, but not of locomotor ataxia. Progressive muscular atrophy in its earlier stages may be mistaken for locomotor ataxia. The wasting of the muscles in anomalous cases may be imperceptible, and the unsteadiness of the individual may alone attract attention. This, with the pain, may raise a doubt as to the true nature of the malady. Hysterical ataxia, such as has been described by Webb, as a rule, is not symptomatized by pain, and the ataxia is not genuine. Syphilis, in some of its forms, also occasionally produces symptoms which are very much like those of this disease; and there may be paralysis of cranial nerves, with pain over the tibia, which may be misleading, when in reality no spinal disease exists.

Chauvët,<sup>2</sup> in his excellent article upon the influence of syphilis in the genesis of nervous disease, dwells upon the connection of syphilis with locomotor ataxia, and quotes many authors to show that the co-existence of these two diseases is a pure coincidence.

In a table showing their relation, eighty-five cases of ataxia are presented :

| Reporter.            | Syphilitic Patients. |                 | Ataxics. |
|----------------------|----------------------|-----------------|----------|
| Fournier . . . . .   | 24 . . . . .         | among . . . . . | 30       |
| Vulpian . . . . .    | 15 . . . . .         | " . . . . .     | 20       |
| Féréol . . . . .     | 6 . . . . .          | " . . . . .     | 11       |
| Siredey . . . . .    | 6-8 . . . . .        | " . . . . .     | 10       |
| Caizergues . . . . . | 8 . . . . .          | " . . . . .     | 14       |

<sup>1</sup>Op. cit., vol. ii. p. 683.

<sup>2</sup> Influence de la Syphilis sur les Maladies du Système Nerveux Central, p. 53. Paris, 1880.

His conclusion is that syphilis has nothing to do with the actual development of primitive sclerosis of the posterior columns, but the presence of syphilitic deposit in this region may undoubtedly give rise to symptoms closely resembling those of the uncomplicated disease.

Buzzard holds, however, that in nearly all cases of locomotor ataxia, either that some remote or recent history of syphilis is disclosed.

**Prognosis.**—Among the number of cases reported by various observers, I have not found many well-authenticated cures. An interesting fact, however, has been observed by Gowers, who states that in the cases of this disease he has seen—and they were a great many—that in families, those persons who reached the age of twenty-five without showing symptoms are exempt, although other members of the same family may have been affected. So important does he consider this fact that in one family in which there were three members affected, he recommended the application of a fourth member who presented himself as an applicant for a life-insurance policy. In regard to this question of age, it must be admitted that it is often a most difficult matter to say when the disease began, for the early pains are mistaken for other troubles. The following table gives, besides other facts, the ages and sexes of eight individuals affected. And it will be noticed that the disease began in these cases as follows: 37, 41, 40, 32, 45, 55, 36 and 42. It is barely possible that in some of these cases the first stage was not characterized by pain intense enough to engage the patient's attention.

AN ANALYSIS OF EIGHT CASES OF LOCOMOTOR ATAXIA AT THE HOSPITAL FOR EPILEPTICS AND PARALYTICS, NEW YORK CITY.

| No. | Sex. | Age. | Duration. | Probable Cause.        | Ataxic Members. | Location and Character of Pain. | Tendon-Reflex.              | Disturbance of Surface Sensation. | Ocular Symptoms.                           | Cerebral Symptoms.                |
|-----|------|------|-----------|------------------------|-----------------|---------------------------------|-----------------------------|-----------------------------------|--|-----------------------------------|
| 1   | M    | 45   | 8 years.  | Syphilis and exposure. | Legs.           | Back and thighs.                | Absent.                     | Anæsthesia                        | None.                                      | Vertigo.                          |
| 2   | M    | 53   | 12 "      | Unknown.               | Legs and arms.  | Arms, legs, viscera.            | "                           | "                                 | None.                                      | None.                             |
| 3   | M    | 54   | 14 "      | Excessive venery.      | Legs and arms.  | Legs.                           | "                           | "                                 | Atrophy of optic nerve.                    | "                                 |
| 4   | F    | 52   | 20 "      | Unknown.               | Legs and arms.  | Back, legs.                     | "                           | "                                 | Atrophy of optic nerve.                    | Vertigo and epilepsy.             |
| 5   | M    | 59   | 14 "      | "                      | Legs.           | " "                             | Increased to marked degree. | "                                 | Atrophy of optic nerve.                    | Occasional epileptic attacks.     |
| 6   | M    | 57   | 2 "       | "                      | Legs and arms.  | Legs.                           | Well Marked.                | "                                 | Normal.                                    | None.                             |
| 7   | M    | 40   | 4 "       | "                      | Legs and arms.  | "                               | Increased.                  | "                                 | Impaired vision.                           | Frontal headache (a coincidence?) |
| 8   | M    | 59   | 17 "      | Intemperance.          | Legs and arms.  | "                               | Increased.                  | "                                 | Dimness of vision due to atrophy of disks. | None.                             |

A peculiarity of the disease is the long intervals of improvement which occasionally occur; and the disease may be stationary for years, but this is very rarely the case. I know of two cases which were so much improved, and remained so well for three or four years, that I flattered myself that I had cured them, but I have since seen a change for the worse in both patients. Balfour<sup>1</sup> presented a case of locomotor ataxia which he claims to have cured. Pollard<sup>2</sup> reports a case which began rather suddenly, and disappeared quite rapidly under treatment. Vidal,<sup>3</sup> Duqueit,<sup>4</sup> and Herschell,<sup>5</sup> all report cures. Vidal's patient, a man of 45, recovered in three months, and Duqueit's and Herschell's cases I consider doubtful as regards diagnosis.

**Treatment.**—From the very nature of the disease, the treatment must be empirical. Nitrate of silver has been recommended by Wunderlich, Charcot, Vulpian, and others, and has enjoyed great popularity as a remedy. Balfour, already alluded to, states that he cured a patient in three months by half-grain doses of this salt repeated three times a day, and by the use of a foot-bath in which a quantity of common salt had been thrown. The feet were also submitted to the influence of a faradic current passed through the water by proper appliances. The salts of silver may be used with considerable impunity without discoloring the skin, and an unnecessary degree of timidity has been shown in their employment. It is well, however, to begin with a quarter-grain dose, and it may be increased to a half, or even a grain, thrice daily.<sup>6</sup> One case of my own was greatly benefited by this drug in combination with *nux vomica*. I have lately tried the phosphate of silver in one-third of a grain doses, with great success, and prefer it to the nitrate. In administering the silver salts, it is well to give them continuously for several months, and then permit an interval to elapse before beginning again. In the early stages of the disease, I prefer the fluid extract of ergot, either in combination with the bromide of sodium or alone. It certainly seems to control the pain. For this purpose a simple remedy often affords great relief. If a few drops of the bi-sulphide of carbon are placed upon a piece of cotton in the bottom of a wide-mouth bottle, and the same be held for a few minutes over the painful spot, great ease will be obtained. Large doses of salicylic acid have an anodyne effect. Among the more efficacious remedies to which I may allude is the sulphur bath, which is too little used at the present day, but has been praised by the French writers especially.<sup>7</sup> It seems to possess, in some cases, powers that are

<sup>1</sup> Brit. Med. Journal, 1875.

<sup>2</sup> Lancet, 1872, vol. i., p. 437.

<sup>3</sup> Gaz. des Hôp., 127, 1862.

<sup>4</sup> L'Union, 122, 1862.

<sup>5</sup> Bullétin Gen. de Thérapeutique, lxiii., Oct., 1862.

<sup>6</sup> De l'emploi du nitrate d'argent dans le traitement de l'ataxie progressive. Bull. Gén. de Thér., 1862.

<sup>7</sup> It has acted wonderfully in cases even of long standing, and deserves a faithful trial.



almost marvellous. A small lump of sulphide of potassium is to be thrown into the tub in which the patient bathes, after which he is to be thoroughly rubbed. In regard to electricity, Meyer has reported several cures by the galvanic current. Onimus has used the inverse current, and I believe has done some good. The indication seems to be that the positive pole should be placed over the painful point, if one can be found, and the negative above. These cases in which cures have been wrought were, I infer, ataxic conditions of a functional character. Faradization of the muscles of the legs and thighs seems to comfort the patient more than anything else. Duchenne thinks that the muscular anæsthesia is benefited greatly by its use, and that co-ordination is improved. Dr. Drinkhard, of Washington,<sup>1</sup> suggested that strychnine injected hypodermically, is a remedy which should not be lost sight of. In one case it promptly relieved the pain. He, however, compares the dangerous appetite of possible formation to that which grows out of the medicinal use of large doses of opium, and fears such trouble. I have used the actual cautery to the spine quite frequently, and have found that constant revulsive effect kept up for some weeks not only diminished the pains, but really improved locomotion. It should be applied down the whole length of the back, on either side of the spinous processes; and, after the epidermis has shrivelled off, subsequent applications are to be made. Belladonna and turpentine internally are recommended by Trousseau, and not only relieve the pain, but seem to help any vesical trouble that there may be. Should we suspect syphilis, the iodide of potassium will be indicated, and a saturated solution should be prepared, and given in increasing doses till forty or fifty grains are taken three times a day. Above all, it must be remembered that nutritious food, cod-liver oil, and moderate stimulation are perhaps more important than medication. I have observed the necessity for quiet and rest. Prolonged muscular exercise is bad, and drives are to be preferred to walking. The patient should seek a warm climate, for this disease is affected by damp, cold weather, very much as is phthisis, and a cold winter always tells upon the patient. The pains also are aggravated by cold and sudden changes, and I find Florida or other southern states to be the most comfortable places for these invalids. Much benefit has been derived from the dark room treatment, and I saw one gentleman who had been greatly improved by a few months of bed-rest in a dark chamber.

Nerve-stretching has been tried in this disease with some apparent success, especially by Langenbeck; but though two-thirds of the reported cases were helped, there was usually a relapse.

Dissipation thwarts any chance of success, and late hours or a debauch will produce a relapse sometimes after encouraging improvement has taken place. Sexual indulgence (when it is possible) is likewise to be interdicted.

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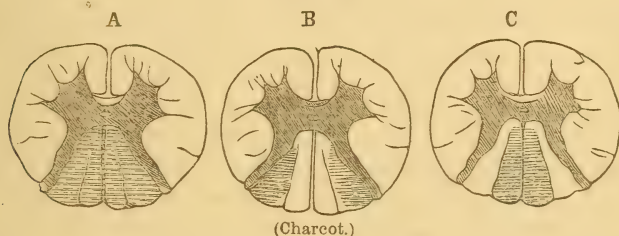
<sup>1</sup> Am. Jour. Med. Sciences, July, 1873.

## SCLEROSIS OF THE COLUMNS OF GOLL.

*(Ascending Degeneration of Posterior Columns.)*

The localization of myelitis in this part of the spinal cord is a matter of great difficulty. Charcot has studied the appearance of degenerative changes in connection with locomotor ataxia, and has found that when limited disease of the columns of Goll was found, the symptoms were those of ascending trouble. In his last work (1880) upon localization, he has presented illustrations which show the invasion of the disease process and its significance.

Fig. 52.



A. Total sclerosis of the posterior columns (columns of Goll and posterior root-zones), *ordinary locomotor ataxia*.

B. Sclerosis of the two posterior root-zones (columns of Goll exempt), *locomotor ataxia*.

C. Sclerosis limited to the columns of Goll—*ascending degeneration*.

Cases of degeneration of the columns of Goll are cited by Charcot,<sup>1</sup> Erb,<sup>2</sup> Simon and Lange. In all of those of the first writer the disease began below, and in fact the German investigators agree the disease begins as a rule by tumors or other forms of disease in the region of the chorda equina, and while at this inferior part it may result in a quite transverse myelitis, it extends upwards, being limited to the columns of Goll. This is beautifully seen in the upper part of the cord, where the degeneration may be well defined. The disease resulting from a transverse myelitis may be transmitted upwards, and degeneration of the columns of Goll be found to extend as far as the floor of the fourth ventricle.

Disease *beginning* at a higher level is very apt to be complicated with a morbid extension into the adjacent parts, so that the appearance figured in Plate A results, and this is explained by the arrangement of commissural fibres found in this part of the cord.

In nearly all cases it is impossible to make a diagnosis between the limited disease of the posterior columns and that which constitutes true "locomotor ataxia." In the cases of Charcot and Pierret the symptoms

<sup>1</sup> *Lçons sur les localisation*, p. 259, et seq., Paris, 1880.

Article in Ziemssen's *Cyclopædia*, p. 773, vol. xiii., Am. Trans.

differ but little from those of the latter disease. It would appear that the success of our diagnosis should depend upon the recognition of irregularity in the appearance of symptoms, the absence of vertigo and ocular trouble; and the predominance of other symptoms rather than the acute pains, which suggest disturbance more of the root-zones than any other part of the cord. Pierret<sup>1</sup> has found the waist constricting band (paræsthesia), unsteadiness when the eyes were closed, and impaired power of preserving the equilibrium, but none of the striking symptoms of locomotor ataxia, in a case of uncomplicated disease of the columns of Goll.

### ANTERO-LATERAL SPINAL SCLEROSIS.<sup>2</sup>

**Synonym.**—Amyotrophic lateral spinal sclerosis (Charcot).

When the anterior tract of gray matter and the lateral columns of the cord are conjointly the seat of the destructive changes, we find permanent contractures following loss of muscular power in both upper and lower extremities, together with extensive atrophy and subsequent bulbar symptoms.

**Symptoms.**—The disease begins without fever; with loss of power in the muscles of the upper extremities, which becomes quite marked after a short space of time, and then follows a *general* atrophy of the muscles of the paralyzed members. In this way the malady differs from progressive muscular atrophy, in which one group of muscles, or even a single muscle, becomes atrophied before others, and in advance of any paralysis. Charcot calls this wasting process “*atrophie en masse*.” Attendant upon the paralysis are deformities, and these are highly characteristic of the disease, and result commonly from contractures of muscles which are less paralyzed than others, so that the stronger muscles overcome the weaker. The flexors of the hands are commonly affected, and these members are flexed and distorted, the fingers being drawn up so that their ends press into the palms, as is the case in other forms of post-paralytic contractures. The arm may be adducted to the side, and forcible adduction or extension is impossible. Pain is usually produced by any violent effort made to overcome the deformity, and the physician is obliged to desist. The patients are able, though their muscles are paralyzed and contracted, to perform certain limited movements, but the same tremor takes place which we observe in other forms of sclerosis when a voluntary effort of any kind is made. In the late stages the emaciation is complete, and the appearance of the hands resembles that seen in progressive muscular atrophy. There are the elevated thenar eminences and the flat forearms, but the limb is still contracted. Charcot alludes to a condition which sometimes affects the muscles of the neck, so that they are contracted to such a degree that the head is fixed and immovable. He relates

<sup>1</sup> Archives de Physiologie, etc., 1873, p. 74.

<sup>2</sup> I prefer this compound title, as it obviates confusion and more definitely expresses the seat of the disease.



a case where the muscles of the inferior maxilla were so contracted as to greatly interfere with mastication.

The progress of the disease is marked by involvement of the tongue, and later by the destruction of the nuclei of the several cranial nerves, so that various losses of special function rapidly follow, and death terminates the patient's sufferings. The inferior extremities are paralyzed in their turn, and are the seat of contractures which resemble in some respects those of the upper extremities, so that his condition is one of helplessness. The legs become rigid when he attempts to walk, and are agitated by tremors so that he is obliged to desist. The contractures in the lower extremities are much more marked than in the upper, and when finally the victim seeks his bed he presents a most abject and pitiable appearance, the legs being twisted and contracted so that he requires the services of an attendant, as he is utterly unable to do anything for himself.<sup>1</sup> Fibrillary tumors may be present just as in progressive muscular atrophy, but are not so constant as in the latter disease. The symptoms which usually herald the approaching end of the disease are those which indicate invasion of the bulb. Paralysis and atrophy of the tongue, vermicular movements of that organ, and affections of speech, are among these, and the orbicularis oris and facial muscles are next attacked, when there may be drooling of saliva and other indications of bulbar degeneration. In short, the symptoms are very much like those of bulbar paralysis. Sooner or later the pneumogastrics are implicated, and death follows. The disease runs its course in from six months to three years.

I have been so fortunate as to see one case of this disease, the note of which I append.

E. S., laborer. About one year ago he noticed an awkwardness in holding his spade, and when engaged in the excavation of a cellar he was unable to throw up the dirt, and at the same time felt unpleasant formication and cramps. These became so distressing that he applied liniments to his wrist and arms, but without any relief whatever. He consulted a medical man, who tried electricity, with no good effect, and after passing two or three months without treatment, he came to me, and I was able to make a diagnosis almost immediately. Both hands were strongly flexed, and the muscles were greatly atrophied. The index finger of the left hand alone escaped contraction. There was some rigid contraction of the forearms, while the arm was carried upwards and forwards by the muscles of the shoulder and thorax, and there was no movement of the elbow or wrist. Fibrillary contractions were observable in the triceps, pectoralis major, and biceps. When I endeavored to straighten the arm he suffered great pain, and begged me to desist. There seemed to be no involvement of the lower extremities, and the patient walked without embarrassment.

Seeligmuller<sup>2</sup> saw several curious cases, which were not only valuable

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<sup>1</sup> There is never cutaneous anæsthesia, the bladder and rectum are not affected, and there is no tendency to bedsores (Charcot.)

<sup>2</sup> Deutsche Medicinische Woch., April 22 and 29, 1876.

as instances of heredity, but which illustrated the course of the disease.<sup>1</sup>

The cases came under the observation of Seeligmuller in January, 1876. The family history, which was carefully inquired into, was remarkably good, with one significant exception—that the parents were first cousins. There was no evidence of syphilis. Seven children—six girls and one boy—were the result of the marriage. Of these, the eldest, aged eleven, was quite healthy; the second, aged ten, was in an advanced stage of the disease; the third was, if anything, worse still, but was not seen; the fourth, a boy, aged six years and nine months, was in the middle stage; the fifth and sixth were healthy; and the seventh, aged one year and nine months, was in the first stage of the affection. The disease began in a similar way in all. Strong and healthy when born, they continued so up to the age of about nine months, when a change took place. Able previously to sit up without trouble, they began to lose this power, and would fall to one or other side; later, the head and chest sank forward. At the age of two years attempts were made to teach them to walk, but their efforts resembled those of an infant six months old. This was exemplified in the youngest patient, who, when supported under the armpits, made jumping movements, the legs being raised from the ground simultaneously. Subsequently the children learned to support themselves with difficulty against a chair, but even this power was lost again. The boy had lately been rapidly losing ground in this respect; he could still, however, drag himself about in his bed, and, by means of a specially constructed chair on wheels, could walk. The two eldest children, when supported in the upright position, could not put one foot before the other; even when lying down, they were unable to move, the upper extremity being useless as supports. The youngest girl could sit for a short time on the table, but cried all the time, and soon fell to one side; she sat with her head and chest inclined forwards, the spine equally curved, and the thighs greatly abducted; when on the lap, however, she could move her arms and legs in all directions.

Contractions at the joints were present in a high degree in the three eldest. In the eldest girl the hands were adducted and pronated; pain was produced by attempts at passive supination, and the hand, when released, jerked back to its old position. The fingers were rolled in towards the palm, but she could still extend them, though very gradually and with great difficulty. The grasp was still perceptible; the right better than the left. The elbows were slightly bent, and nearly fixed. The knees were half flexed, but could, with great force, be moderately extended or flexed still more, though on leaving them they sprang back with a jerk. The feet were in the position of advanced equino-varus; the tendines Achillis were perfectly rigid. All attempts at passive movement produced considerable pain. The boy was put under the complete influence of chloroform, and the rigidity of the joints then so increased that the whole body could be raised from one leg and held out like a piece of wood. The youngest girl has so far no contractions.

Atrophy of the muscles was marked in the two eldest under observation. With the exception of those of the face, it was evenly spread over the whole system. The wasting in the case of the girl was considerable, so that the head seemed too large for the attenuated neck, and was moreover unsteady. The parents were confident that in all three the

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<sup>1</sup> London Medical Record, June 15, 1876.

wasting was not visible for some time after the loss of power showed itself.

In the eldest child the reaction of the tibial and peroneal nerves was normal with both currents; but the irritability of the muscles was decidedly lowered everywhere. Of those on the back of the forearm, the supinator longus alone responded promptly. In the youngest girl, faradic excitability of both nerves and muscles was perceptibly lowered in all extremities, but especially in the left lower. Galvanic excitability was lowered in the same way, and in the tibial nerves was almost *nil*. Ordinary reflex irritability not increased. That of the tendons, however, was present in a high degree in all. Fibrillary contractions were markedly present in the eldest girl, and could be produced by simply blowing on the skin. Sensibility was normal in all.

Of the symptoms noticed by the parents, that which made its appearance last was the gradual loss of the power of speech. Thus, in the two eldest girls, this was tolerable until their sixth year, when it became less and less distinct, until finally only inarticulate nasal noises could be made. In the girl, the lips, soft palate, and uvula were all paralyzed, and the tongue lay in the mouth like a mass of dead flesh; its tip could be advanced only as far as the teeth. In the boy the same symptoms were present, but in a somewhat less degree. The youngest child could say a few words, but these had a slightly nasal tone. Swallowing in the two eldest girls was difficult; in the boy, tolerable. The form of the skull was unusual in all, but especially so in the eldest. It was very broad between the parietal eminences, and very undeveloped in the frontal region. The forehead was low, and the head appeared altogether too small for the face. In the eldest girl the features were coarse; the expression was vacant, but usually amiable; the pupils were much dilated; the saliva flowed continuously out of the half-opened mouth; and, indeed, her general appearance was that of an idiot; though, in point of fact, the intellect was very fairly developed. The faradic excitability of the facial muscles was decidedly increased; the galvanic was normal.

**Causes.**—No definite causes are known, though exposure is believed to have much to do with its origin, and Charcot's cases are thus accounted for; but we may also consider that dissipation and hereditary influences play an important part in the etiology of the affection. It is a disease which rarely occurs before adult life, so far as we are enabled to judge from the limited number of cases which have been reported.

**Morbid Anatomy.**—To Charcot belongs the credit of having made the distinction between progressive muscular atrophy and lateral amyotrophic sclerosis. Previous to 1867, examples of this affection were considered to be cases of progressive atrophy, which were anomalous in the fact that the lateral columns were affected. Jaccoud<sup>1</sup> considers the sclerosis as *circumscribed* or *diffused*. Like sclerosis in other regions, the tissue-changes may be observed with the naked eye, either invading the white or the gray matter separately, or more often together. In this case the lesions are of ancient date. The connective tissue is firm and shrunken, and the color of the hardened spot is gray or pinkish-gray. The meninges may be adherent

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<sup>1</sup> Op. cit., p. 319.



to the cord if the sclerosis be circumferential, but it is more common in uncomplicated sclerosis to find no such change. The microscopical appearances are like those seen in locomotor ataxia, as the character of the lesion is identical, the only point of difference being the location of the tissue-change. *Circumscribed* sclerosis is more rare than the diffused variety, and few cases have been observed. Of examples referred to by Jaccoud, in one the lesion was confined to the lumbar enlargement, and invaded the entire anterior columns and a part of the lateral columns; and in another, in which the autopsy was made by Frommann,<sup>1</sup> "the sclerosis occupied the lumbar segment and the inferior portion of the dorsal region. It involved in different degrees all the white matter, and the gray was not affected except in the gelatinous substance and in the parts of the posterior cornua which bounded the lateral column." The sclerosis has involved the entire antero-lateral columns, the anterior columns alone, or the lateral and the lateral and posterior conjointly. In diffused sclerosis, nodules are found in various parts of the brain and cord, but the predominance of the sclerosis in the antero-lateral column gives prominence to the symptoms which I have described.

**Diagnosis.**—It is possible that this disease may be confounded with either progressive muscular atrophy, lateral sclerosis, or spinal paralysis. In the first we find a train of symptoms consisting of neuralgic pains, atrophy of single muscles or groups, and involvement of other muscles progressively, and secondary paralysis. There are besides no spasmodic contractions. In lateral sclerosis there is no atrophy beyond that resulting from inaction. In the disease known as spinal paralysis the lower extremities are generally affected first, and reflex excitability and electric irritability are diminished, which is not the case in the disease which has just been described.

**Prognosis.**—About as bad as it can be, though very few cases have been reported. It would seem that there should be as much chance in this disease as in lateral sclerosis, which is sometimes cured, but such is not the case.

**Treatment.**—I think it may be said that no treatment offers any real assurance of success.

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<sup>1</sup> Anatomie des Rückenmarks, Jena, 1864.

## CHAPTER XII.

## DISEASES OF THE SPINAL CORD—(CONTINUED).

## DISEASES OF THE LATERAL COLUMNS OF THE SPINAL CORD.

THE various forms of disease of this part of the spinal cord may be tabulated with reference to their symptom significance as: 1. Infantile spastic paralysis (*spastische spinallähmung* of Erb.<sup>1</sup>) 2. Functional spastic paralysis (*Störungen-neurosis* of Berger.<sup>2</sup>) 3. Hysterical spasmodic paralysis. 4. <sup>3</sup> Adult spasmodic spinal paralysis (Primary symmetrical lateral sclerosis of Charcot.)

1. Is of course an affection present at birth, or commencing very soon after, and has continued through life in all the cases so far observed.
2. Is not confined to any age, but so far the reported cases have been among adults. It has its analogue in functional paralysis and disturbances of sensation dependent upon ischæmia of other parts of the cord.
3. A disease of adult life, and so far has been seen only among women.
4. A disease of adult life, rarely beginning before the twelfth year, and sometimes curable.

**Symptomatology.**—The positive symptoms of lateral column disease may be enumerated as paresis, with rigidity and contractures, and increase of all forms of reflex excitability, and especially that of the tendons.

Of the negative symptoms we speak of the absence of atrophy, and bladder and rectal complications as well as true ataxia, and it may be stated that cerebral symptoms are never present.

In the various forms of lateral disease, there is great irregularity in the loss of power, either in extent or period. In the infantile cases it may date from earliest life, and only be recognized at the time when the child is naturally expected to walk; or it may gradually occur later in life as the initial stage of the disease. This rule holds good in every case; for in the examples of secondary trouble there is always an early paresis even though there may be preceding anæsthesia

<sup>1</sup> Memorab. Monatsschaft, f. r. p. a. xii. Jahr. 12 H. 1877, p. 529.

<sup>2</sup> Centralblatt, 1878, p. 13.

<sup>3</sup> Séguin, Strumpel \* and others inclined to think that spasmodic spinal paralysis may be produced by a variety of lesions among which are compression myelitis, tumor and cerebro-spinal sclerosis. This is undoubtedly true to a certain extent but it must be acknowledged that the spastic paralysis thus induced is seldom uncomplicated, and that sensory and other irregular symptoms are produced as well.

\* Archiv. für Psychiatrie, x. p. 676, and xi. p. 27.

or other sensory troubles. The early signs of impaired power are manifested in a variety of ways: the individual easily tires; and a short walk produces a sense of fatigue referred to in the flexure of the knees. He leaves his bed with difficulty, and his legs are used awkwardly; and as the day advances he feels more disinclined to walk or move about. Should the upper extremities be those first affected, he finds himself unable to grasp his tools as forcibly as he once did. If he is a clerk, his pen is used clumsily and its point is not kept in contact with the paper, but traverses the lines unsteadily, so that the writing is exceedingly tremulous and without character. The paresis becomes more decided, and is connected with spastic rigidity. Later on, as it grows more profound, it resembles, to some extent, certain well-known forms of paralysis—but there is no anæsthesia.

This similarity is very decided in the hemiplegic forms, but the loss of power, however, is likely to affect the different members in a decidedly irregular manner, perhaps appearing in one leg first, then the other, and finally the arms; or it may affect one leg, then the arms of the same side, and then those of the other side. The limbs may be the seat of paresis, which varies on both sides in profundity. Although sclerosis of the lateral columns on one side only giving rise to a hemiplegia of spinal origin (such as have been especially alluded to by Berger), may occasionally occur, it will be seen, from an inspection of reported cases, that in primary disease of the lateral columns, and even in the transverse varieties of secondary degeneration, that the paresis is paraplegiform. The paresis is suggestive of extensor paralysis; and in supine posture in the advanced stages, the patient is usually unable to raise his heels more than four or five inches from the surface upon which he may be lying, and in most cases not even to this extent. Combined with the paresis is a certain amount of rigidity, which exists in every case, and varies from a simple spastic condition to one attended by absolute contractures. The paresis and rigidity, gradual in their method of appearance, are rarely universal; but in nearly every case of either primary or secondary disease, ultimately affect both extremities. The earliest evidences of motor irritation are shown in the muscles of the lower extremities, notably in a certain spastic stiffness of those of the calf and of the posterior and inner aspects of the thighs, and as a result of this trouble, there is great rigidity where passive movements of the knee and ankle joints are made; and when any attempts at locomotion or other movements requiring use of the feet are essayed, these members become extended and quite rigid. This rigidity, like the tendon reflex, seems to be increased by warmth (though in a case reported by Kussmaul the reverse was observed), and it is especially troublesome when the upright position is assumed. When the knee is bent and the leg flexed, it will be found that the hamstring tendons stand out as rigid cords, while there is more or less resistance to flexion of this kind. The gait of patients suffering from disease of the lateral columns, has been called by the Germans “spastichergang;” and its peculiarity depends upon the combination of paresis and muscular rigidity—the latter being increased by the act of



putting the foot to the ground. In the beginning, as a result of the loss of power, the patient constantly stubs his toes, which comes in contact with any little elevation which may be in the floor or surface upon which he walks. Afterwards the embarrassment is increased by the spasms which involve the muscles upon the posterior aspect of the leg; and there occurs a species of talipes equinus, the toes, however, being usually flexed. The patient, from the first, walks with difficulty, his feet becoming interlocked and entangled, and through a rigid contraction of the thighs, the knees are brought together; and as a result of friction these internal surfaces will be found to be callous and roughened. The knees are often *sunken*, so that the anterior leg or thigh surfaces form almost an obtuse angle, and in the advanced forms of disease of this kind, these deformities of extension and adduction become very conspicuous, and the patient becomes so helpless that he requires a cane or crutches.

In the upper extremities deformities and spastic rigidity are neither so markedly or constantly shown, although in rare cases terrible distortions of the variety described by Charcot<sup>1</sup> and Strauss<sup>2</sup> are sometimes seen.

As a later result of continued and persistent contractions of the muscles ending in the tendo-achillis, and in other tendons, there may result conditions either of talipes equinus, valgus and varus, and the patient's efforts to walk cause him very great distress, as his weight comes upon his distorted foot.

A peculiar deformity, first noticed by Charcot,<sup>3</sup> and which I have several times observed,<sup>4</sup> is the abdominal contraction which gives rise to a very pronounced anterior curvature of the body; and, as a result, there is a protrudent abdomen and a deep fissure below the lower border of the ribs. In such cases there is usually some local wasting of the muscles of the back, just as there would be in any muscles subjected to disease, and kept upon a stretch for a long period of time, but in no respect is there true atrophy from deficient central innervation. The head is never affected by motor trouble; and there is no paresis of the muscles of the neck.

One of the marked distinguishing features of disease of the lateral columns, is an exaggeration of reflex action which is evinced in several ways. Not only is the skin reflex increased to a decided extent, so that tickling, simple contact of the clothing, or even blowing upon the surface, will provoke variations of motility of irregular and disorderly character, but the "tendon-reflex," which plays an important part in all these cases, is excited. There are a number of manifestations of motor irritation which have been described independently; but I am of the opinion that they all resemble each other, and all depend on activity of the so-called "tendon-reflex." The so-called Knie or Unterschenkel Phänomen and Füss Phänomen of Erb and Westphal, are simply varieties of

<sup>1</sup> Leçons sur les Maladies du syst., N., 1872-3.

<sup>2</sup> Op. cit., p. 16.

<sup>3</sup> Leçons sur les maladies du syst., N. 1878.

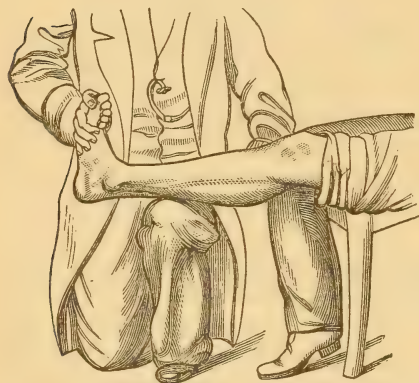
<sup>4</sup> New York Medical Record, Oct. 28, 1878, p. 323.

chronic movements which follow forcible stretching of different tendons when the knee and ankle joints are bent in flexion, and are varieties of tendinous reflex. The simplest and usually most easily produced movements follow flexion of the foot.

From an inspection of a large number of cases, I am certain that the value of this test depends very much upon the degree of flexion; for if too little stretching of the tendo-achillis is made, the results will be as unsatisfactory as when this tendon is over-tensely drawn.

To evolve this clonic movement (dorsalklonus of the Germans; trepidation provoquée of Charcot), the operation is to grasp the leg (but not too tightly) with the left hand, while the palm of the right hand is brought in apposition with the plantar surface of the patient's foot, which

Fig. 53.



Method of Provoking Dorsal Clonus.  
(GOWERS.)

is passively flexed, so that the toes are forced *slightly* upwards. The foot is kept in this position, and usually in a very short space of time, often immediately, there is manifested a clonic spasmodic agitation of alternate flexion and extension.

Sometimes such motor disturbance continues after the hand is removed, the patient's foot being extended, the heel retracted by the muscles uniting in the tendon achillis; and while raised several inches from the floor, it is agitated for some time,—several

seconds usually, but I have seen cases in which the trepidation lasted nearly half a minute. This trepidation is extremely variable; and, like the movements following the tapping of the tendon, it presents different features in different cases and at various times. In some cases, it instantly follows the original stimulation, and increases in frequency, the intervals between the separate contractions decreasing, and the muscular movements increasing in violence.

In one patient at present under observation, the initial tap causes at first an immediate but not very violent *kick*. This is followed by others which increase in the frequency of their appearance seemingly as if every muscular contraction arouses new collections of nerve force and promotes the escape of nervous discharges, until finally as the irritability of the central apparatus becomes exhausted, the contractions grow weaker and ultimately cease. In some cases the simple passage of the finger over the skin of the foot will give rise to the epileptoid tremor, and Joffroy<sup>1</sup> has

<sup>1</sup> Gazette Médicale de Paris, 1875, Nos. 33-35.

repeatedly produced the trepidation by the application of such gentle excitants to the skin as the contact of a finger-tip or a damp compress.

Grasset<sup>1</sup> reminds us that when the patient is under emotional excitement, or when he makes an effort to execute certain movements; or, again, when embarrassed at meeting a strange person, clonic spasms are sometimes spontaneously produced.

A variety of clonus, called by the French "*trepidation spontanéé*," takes place when no apparently affecting stimulation is used. The movements of a tremulous character which agitate the lower extremities of a healthy person, who is fatigued after a long walk, or some such effort, is but a simple illustration of the condition of affairs which exists in disease of the lateral columns, in a more pronounced degree. A constrained position, or one in which the tendons are slightly stretched, is highly favorable to the causation of a paroxysm of tremor, and where the central irritability is great, the mere contact of the clothing is oftentimes all that is required as a peripheral irritation. The recumbent position and rest seem to modify the violence and frequency of these phenomena; for it is only in exceptional instances that they occur during sleep. As soon, however, as the feet come in contact with the ground, the retraction of the heel takes place, and every step in walking is connected with more or less spasmodic movement.

A form of reflex trouble which has received but little notice, is the abdominal reflex. This I have noticed in lateral disease, and I think it should be considered always as a pathognomonic sign of the affection. When the finger is passed ever so slightly over the abdominal parietes, there will be a peculiar, almost vermicular contraction of the underlying muscles. I have never seen this sign absent in spasmodic spinal paralysis. This excitable condition of the abdominal muscles has probably something to do with the curious action of the bladder; and it is probable that the muscular fibres of this organ are also subject to reflex spasm which results in the forcible and spasmodic discharge of the urine which sometimes occurs.

In certain cases the action of the will is capable of modifying, if not stopping, disorderly movements of a reflex nature; but in the great majority the reverse is the rule, and the attempted exercise of the volition is frequently all that is required to increase the movements.

In one case I have witnessed a phenomenon which is not uncommon in connection with the transmission of peripheral painful impressions—I allude to delayed conduction. In this case the tap was not immediately followed by contraction; but from three to five seconds elapsed before any movement was to be observed.

In pure uncomplicated disease of the posterior part of the lateral columns there should be no muscular atrophy. In varieties beginning with disease of other parts, or injury, such a condition of affairs is possible but not commonly seen. Any loss of muscular substance is simply due to

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<sup>1</sup> *Maladies du Syst., n.*, Paris, 1878, p. 375.



inaction of the limbs, and is of peripheral origin, and involves the entire limb. Bed-sores are not a feature of the paraplegia, at least not until the other parts of the cord become involved; but in the early form of what may be a secondary local affection they are sometimes seen, as was the case in two or three of Séguin's patients. In the latter stage of primary disease they do occur and have been occasionally observed. In no cases have I observed skin diseases, arthropathies, or other indications of defective nutrition. In the confirmed and advanced examples of the disease, a mottled or bluish appearance of the extremities (such as is witnessed in pseudo-hypertrophic and infantile paralysis), is quite common. This is more noticeable when the patient's clothing is removed and the skin exposed to the air, when the pink blush appearing at first gradually assumes a dusky hue.

Although all authorities deny the existence of any form of sensory alteration, they nevertheless prove by their published cases that in the earliest and last stage of disease of this part of the spinal cord, various sensory phenomena are presented. For instance, in seven out of twelve cases of primary disease of the lateral columns, there were either pains, anæsthesia, or light forms of surface hyperæsthesia. "Tingling," or "burning" sensations, dragging pains, "pricking," or "numbness" are spoken of, and probably arise from some irritation of the posterior nerve roots.

It may be stated positively that absence of anything like sensory disturbances, such as are found in other spinal diseases, is the rule; but it cannot be denied that an occasional or early diminution, or more commonly, elevation of the cutaneous sensation, is a feature of affections of this kind.

In the secondary disorders, where perhaps a congestion of the posterior columns is the primary marked process, or where pressure is made by some growth, or, by a diseased vertebra, or, as is sometimes the case, by the products of inflammation in meningitis, there must be more or less disturbance of sensation. In special varieties this is decided, and where associated with hysteria it is not unreasonable to expect to find anæsthesia; but unlike the impaired sensation in true spinal disease, it is irregularly distributed, and often associated with ovarian hyperæsthesia.

In one of the cases reported by Séguin there was anæsthesia, probably the result of injury of nerve tracts other than the lateral columns, but as in other cases the *symptoms of lateral disturbances predominated*.

Tactile sensibility seems to be in no way affected; and appreciation of heat and cold are usually normal, except in advanced stages, when subjective cold is complained of.

There are never any indications of paresis of the bladder or rectum. Constipation is not usual; and if there is any bladder trouble it is one of a sthenic nature, and accompanied by spasmodic ejection of the urine.

The patient is quite able to stand with his eyes closed, before his loss of power renders him helpless—and he can co-ordinate properly. The only exception to this rule is when the disease has involved the posterior columns, as in the complicated cases mentioned by Erb.

## I.

CONGENITAL IMPERFECT DEVELOPMENT OF, OR DEGENERATION OF THE  
LATERAL COLUMNS OF THE SPINAL CORD. INFANTILE  
FORM, "SPASTISCHE SPINALLAHMUNG BIE  
KLEINEN KINDERN" OF ERB.

The subject of spasmodic spinal paralysis of infancy has received but passing notice, and a contribution of Erb's<sup>1</sup> is the only description to be found of the disease which is an analogue of adult spastic paralysis of the primary form. Four cases were presented by this observer, two of which were described in his second article<sup>2</sup> in Virchow's Archives; and two others are detailed in the communication before referred to.

I have seen several cases which are clearly marked examples of spastic infantile paraplegia. Several of these cases have also been observed by others, but not recognized and described as lateral column disease, and in more than one case the disease has been regarded as the result of preputial irritation from phimosis.

The paresis is usually not recognized until a year or so after birth, when the child should walk, but does not do so; and in such cases the ailment, as Erb has pointed out, has too often been mistaken for infantile palsy or some such common disease of early infancy. If this error is not made, ante-natal cerebral hemorrhage or spinal traumatism is generally supposed to account for the paralysis. One-sided brain atrophy, such as has been alluded to by Taylor,<sup>3</sup> produces a hemiplegic condition with contractures, exalted tendon-reflex, etc.; but cerebral symptoms of greater or less importance are added thereto.

Finally, it has been the fashion of late to ascribe all the trouble to an irritated and phimosed prepuce. Circumcision has even been tried in many instances; but the rigidity and paresis have remained the same, for in all of these cases, the trouble was far beyond the surgeon's knife.

In this form of disease, or congenital partial absence of the lateral columns, the contractures, according to Erb, make their appearance at a very early age. In one of the patients I have seen, the limbs are as rigid at the age of seven, as they would be in the advanced stage of this disease in an adult; and in such a condition, I understand, they have been since the third year.

This early development of contractures is ascribed by Erb to the imperfect voluntary power which belongs to childhood, which prevents the little patients from exercising or resisting the advance of the deformity.

Subjective coldness is noticed, and the cutaneous circulation is sluggish, so that the limbs have a mottled appearance. The ability to speak seems to be impaired—not from a condition of *mental* weakness, however, for the mind of many of these children is quite active; but there appears to be both a local awkwardness and a disinclination to talk. Unless the

Op. cit.    <sup>2</sup> Virchow's Archiv., B. 70, 1877, p. 293.    <sup>3</sup> Guy's Hosp. Rep., 1878.

patient is held upright, he is quite unable to walk alone, for there is crossing of the legs, and adduction of the thighs. If a determined effort is made to walk (he being supported meanwhile), the feet will be drawn into the position of talipes, and his toes will catch the ground at every step. The disposition is for the feet to be drawn across each other, so that in an extended position, one foot covers its fellow, and so they remain. When laid upon the bed the legs and thigh are sometimes drawn up and agitated by clonic movements. In severe cases the loss of power is so great that (as in adult cases) the patient cannot lift his feet or raise his legs.

No sensory disturbances are complained of; and in but one of Erb's cases was there any symptom of this kind, and that a slight hyperæsthesia. Skin and tendon reflexes are increased. Bladder and sphincter ani, normal. Cerebral symptoms, *nil*.

A curious fact appears to be established,—and this is, that in three of the seven cases I have collected, the children were prematurely born; and I think great importance of a pathological kind must be attached to such a state of affairs.

## II.

### FUNCTIONAL DISEASE OF THE LATERAL COLUMNS.

The recently reported case seen by Kussmaul<sup>1</sup> is an example of this kind; for the favorable results obtained by him were highly suggestive of such a conclusion. Berger<sup>2</sup> has also seen a case; and I have no doubt but that many of the cases of spasmodic troubles of the lower extremities, known heretofore as “functional spasms,” are after all only varieties of ischæmia of the lateral columns.

In Hanfield Jones' work,<sup>3</sup> I find reference to a case reported by Baumberger, which is as follows:—

The patient was a youth, 19 years of age, who during convalescence from pneumonia, began to suffer with a spasmodic affection of the lower extremities. “As soon as he touched the ground with his feet, all the muscles of the lower extremities fell into a state of tetanic rigidity, interrupted by the most violent, sudden contractions, which threw the patient upwards; and during their rapid recurrence increased in intensity, so that the patient had to be supported. At the same time, the face was flushed and distorted, the pulse accelerated and extremely feeble. The moment that the patient sat or laid down, all the movements ceased. If, while lying in bed, the soles of his feet were pressed, the same phenomena

<sup>1</sup> Berliner Klin., Wochenschrift, Sept. 23, 1877.

<sup>2</sup> Abst. in Centralblatt, July 13, 1878.

<sup>3</sup> Schmidt's Jahrsbericht, vol. cij., pp. 23–4, and H. Jones on Functional Nervous Disorders, p. 398.



appeared, but with much less intensity." He was cured by sedatives and cold affusions.

In the interesting case reported by Kussmaul, complete recovery took place within less than one year.

### III.

#### HYSTERICAL SPASMODIC SPINAL PARALYSIS.

The celebrated case reported by Charcot of hysteria, in which the four extremities were contracted, is one which illustrates a form of disease of the lateral columns occurring as an outgrowth of the neurosis which is so commonly thought to be a purely functional affection. This and other cases are so well marked, however, and present such unmistakable symptoms of both diseases that I think a hysterical variety of spasmodic spinal paralysis may be recognized.

In all of the cases to which I shall refer, it is probable that the primary disease was purely peripheral, and as a central degeneration has been known to occur after section of important nerve trunks there is no reason why we should not with perfect reason recognize the same pathological origin in cases where long existing hysterical paralysis has been connected with a more than ordinary inactivity and disuse of a member.<sup>1</sup>

Charcot, in his early fasciculus, (1872-3), goes quite extensively into the question and describes the "tremulation convulsive," and other symptoms. He says: "*Quelle condition est donc survenue et a entretenu ainsi l'existence de cette paraplégie avec rigidité des membres? Evidemment, dans les cas récents de contracture hystérique, la modification organique, quelle qu'elle soit, quelque siège qu'elle occupe, qui produit la rigidité permanente, est très-légère, très-fugace puisque les symptômes qui lui correspondent peuvent disparaître tout-à-coup, sans transition, \* \* \* il s'est produit, à une certaine époque, une lésion scléreuse des cordons latéraux, lésion que la nécroscopie permettrait actuellement de reconnaître.*"

Briquét has seen cases of paraplegia complicated with contractures, and mentions three examples. In these cases there was pain and rigidity, especially when passive movements were attempted. One of his cases afterwards fell into Charcot's hands, and is that of which we have spoken.

The development of symptoms indicative of lateral column disease is rarely an early feature, and in the reported cases there was a primary hysterical paralysis which had lasted some years, when the first indications of the degeneration of the lateral columns were shown in an increase in all the reflexes, and an increase of the rigidity of the contracted limbs.

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<sup>1</sup> *Traité clinique et thérapeutique de l'hystérie.* Paris, 1859.

In more than one of Richter's<sup>1</sup> cases there was a decided hysterical element, but this was not exhibited before the more important special symptoms had shown themselves.

#### IV.

##### PRIMARY DEGENERATION OF THE LATERAL COLUMNS.

(*Tabes Dorsalis Spasmodique, Spasmodic Spinal Paralysis, Lateral Spinal Paralysis.*) *Tetanoid Paraplegia (Séguin).*

The disease which by Charcot has been supposed to be essentially a sclerosis of the lateral columns of the spinal cord, though in such a conclusion he has not been supported by Erb, has been called by the former "*Tabes dorsalis spasmodique*,"—and by Erb, "*spasmodic spinal paralysis*."

With the exception of the few infantile cases already referred to, which I do not believe to be always identical with those in which the disease begins later in life, so far as pathology is concerned, the reported cases have all been among adults. In the cases so far observed, the beginning of the disease has been singularly slow and insidious. There has been no febrile stage, and absolutely none of the early and sudden symptoms which attend the development of many of the spinal paralytic diseases; but, on the contrary, the appearance of symptoms has been very gradual.

In most of the cases brought forward, there have been initial symptoms of a sensory character, although few of them have been more than irregular and fugitive. Dragging pains in the hips and down the back of the thighs, pain in the back, and sometimes hyperæsthesia of no very lasting or severe kind, enter the list.

In Erb's<sup>2</sup> cases (16 in number), seven presented sensory symptoms in the first stage. In six the pain was, without doubt, due to spinal irritation; and in the other cases there was a doubt in favor of articular rheumatism. There were various transitory and ill-defined pains, formations in the fingers and soles, and subjective cold. In Schulz's<sup>3</sup> paper, other cases with such initial symptoms are mentioned. Charcot,<sup>4</sup> however, does not believe in the existence of pains during the first stage, and a few other authors agree with him; but in the German and American examples of this disease, so far reported, there is ample reason to believe in their existence in about one-half of the number of cases. These sensory troubles usually last for a few months, and may be coincident with the appearance of muscular weakness, such as has been described under another head.

<sup>1</sup> Deutsches Archiv. für Klin. Med. 18, 6, p. 365.

<sup>2</sup> Virchow's Archiv., Bd. lxx., H. 2, page 24, et seq.

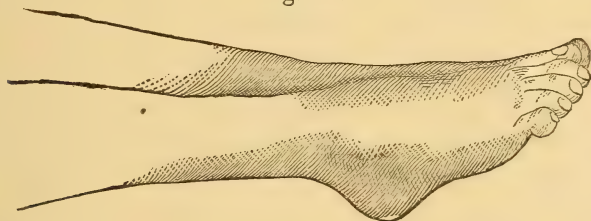
<sup>3</sup> Archiv. der Heilkunde, 1877, page 352.

<sup>4</sup> Leçons sur les Malad. du syst., N. 4<sup>me</sup>. fascic., page 279.

Patients who are in the advanced stage of the disease present in addition to great loss of power, contractures of advanced development; and, as a consequence, there is deformity which is always quite prominent.

As to the loss of power, it will be noticed that in nearly all the reported cases the lower extremities were affected in the beginning, although it is not rare to find either hemiplegic cases, or those beginning on one side and afterward involving the other, this extension occupying a long period of time. Again, the upper extremities are sometimes affected first; but these cases are extremely rare, and I can find but two mentioned. It

Fig. 54.



Contraction of feet in an advanced case of primary degeneration of the Lateral Columns.

however follows that when loss of power begins below, the arms are quite likely to be affected; so that the contractures, trepidations, and all the symptoms already shown below are likely to appear in the upper extremities after two or three years. Even the muscles of the trunk, as shown in one of my cases, are finally implicated.

Bétous makes the third stage of the disease include general contractures of the upper and lower extremities and trunk muscles. The loss of power can hardly be called an absolute paralysis, for the paresis is unequal, and the patient possesses for a long time a great deal of ability to perform certain actions with a great deal of ease, while others are impossible.

Motor irritation is a feature of the second stage of the disease, and accompanies the paresis. The first indication of stiffness marks the appearance of this symptom, and a variety of irregular disorders of motility follow, such as twitching of the feet, tremor amounting almost to clonic spasms when the toes are allowed to touch the floor, and rigidity. when passive movements are made, then other phases of excitement in muscular action are exhibited in different degrees, and at different times until the disease has run an extended course. I have found that in some old cases the clonic movements following excitation are not so active as in the early stages, but that spastic rigidity, and contractures apparently uninfluenced by any ordinary excitation, exist; and also that there is no apparent increase of rigidity in connection with the excitation of any special movements.

As to the negative symptoms of the disease, there is little to be added



more than what has been stated in speaking of general symptomatology. It may be said, however, that there is no impairment of the sexual powers.

The disease ultimately reaches a stationary period; and unless there be a subsequent acute myelitis which ascends and involves the bulb, the patient is likely to live for years, finally to die from an intercurrent disease. After the stationary period is reached he is perhaps helpless, and is confined to his bed. His contractures may become painful, and in general his health suffers through inaction and want of exercise.

In some cases the attempt to stand is attended with great suffering, as the toes are flexed; and when the entire weight of the body is thrown on them in this constrained position, the patient is often unable to progress even with the aid of a stick or crutches without great agony.

I have noticed, in connection with the other symptoms in two of my cases, a great deal of emotional disturbance, which at times amounted to hysteria; and I am inclined to believe that this is but another illustration of the appearance of symptomatic hysteria in connection with organic nervous disorders, such as has been clearly described by Charcot, Séguin and others.

**Causes.**—The causes of disease of the lateral columns are but little known, if we may put out of the question such mechanical factors as external disease or pressure, such as are found in secondary degeneration.

A reference to some of the forms of trouble spoken of in other pages is all that may be necessary under this head (I allude to the hysterical and infantile forms). In the first, I think there can be no doubt as to the origin of the affection as its name implies; while in the other there are actual cavities in the cord; degeneration with syringo-myelia or non-closure of the central canal; or imperfect formation of the lateral columns.

In such cases, there seem to be no hereditary influences to explain their origin except perhaps consanguineous marriages; and we arrive at about the same result when we attempt to trace back influence of this kind in cases of cleft-palate, hair-lip, and congenital deformities of other kinds.

In one of Erb's cases occurring in infancy, the fact that five other children in the same family were born before full term, is suggestive of a tendency to non-development. In Richter's four adult cases, there was a history of insanity on the father's side in two cases, and sclerosis in a third.

An infantile case is reported by Berger, in which the disease followed an attack of diphtheria; but this is the only infantile or adult case in which I can find such a complication, except one, a man who had scarlet fever in early life, which was the beginning of his serious trouble. The lateral columns of the spinal cord are rarely the seat of primary disease until after the twentieth year,—although Erb has reported the disease in a girl of sixteen. In hysterical cases, even, the primary paresis and contractures rarely appear before several years of hys-

terical paralysis have passed. In one of my cases the disease was established at twenty-two; and in none of Charcot's cases did the affection appear before adult life. In secondary disease, there is no regularity in the question of age. I think in the extra-spinal form, childhood is the period when we may expect the causation of such troubles; while if there be tumors, effusions of blood, or meningeal disease, there can be no influence referred to age.

The ages of all the patients with primary disease (spasmodic tabes), whose histories I can gain access to, are the following:—

|                             |    |                             |    |
|-----------------------------|----|-----------------------------|----|
| Between 15 and 20 . . . . . | 2  | Between 40 and 50 . . . . . | 9  |
| “ 20 “ 30 . . . . .         | 8  | “ 50 “ 60 . . . . .         | 3  |
| “ 30 “ 40 . . . . .         | 15 |                             | —  |
|                             |    | Total . . . . .             | 37 |

As to the occupation of these patients,—

|                                  |                                |
|----------------------------------|--------------------------------|
| 3 . . . were . . . laborers.     | 1 . . . was a . . . shoemaker. |
| 2 . . . were . . . peasants.     | 1 . . . “ . . . painter.       |
| 2 . . . were . . . tradesmen.    | 1 . . . “ . . . printer.       |
| 1 . . . was a . . . barber.      | 1 . . . “ . . . butcher.       |
| 1 . . . was a . . . teacher.     | 1 . . . “ . . . carpenter.     |
| 1 . . . was a . . . car-driver.  | 1 . . . was a . . . clerk.     |
| 1 . . . was a . . . silversmith. |                                |

and in twenty cases the occupation was not stated.

Of these patients 22 were men, and 15 women. In fact, the disease is not so common among women, and in many of the cases there was an hysterical element, notably so in the case of the Princess F., reported by Erb. In one of his articles he refers to the fact that the disproportion in sex is not so great as in locomotor ataxia. Climatic influence has been alluded to: in fact the singular circumstance that a number of Erb's cases were from Rheinisch Bavaria led him to think that there was some endemic influence; but the subsequent recognition of cases in all parts of the world proves the contrary.

In one case reported by Bétous and another by myself, the patients were metal workers; but at least in one of these cases there were other causes; so the theory of metallic poisoning must fall to the ground. Syphilis has not entered into the history of the cases; and Erb does not think it has much influence in the production of the disease. “Damp, humid cold,” in the experience of Charcot, who has seen five cases, has existed as a cause; and in many cases, exposure to rain, excessive venery or dissipation have played parts in the development of the disease. So little is known in regard to the genesis of all forms of sclerosis, that any attempt to solve the problem must be speculative. I believe that locomotor ataxia (and probably the disease in question) is undoubtedly due to what is at first but an ischæmic spinal state. In certain individuals of sedentary habits and

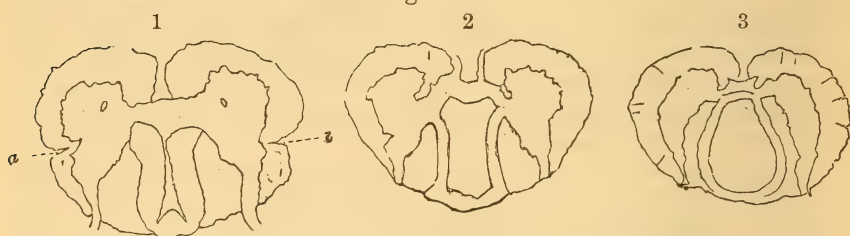
nervous temperament, occasionally the victims of the gouty vice, the cord is subject to sudden modifications in circulation, and consequently in nourishment, and as a result a condition of "spinal irritation" in the primary trouble which may depend upon anæmia on the one hand, or unequal congestion on the other; and as a result of such changes a hypertrophy of the connective tissue follows, which constitutes the sclerosis.

**Pathology and Morbid Anatomy.**—The proper discussion of the genesis of congenital spastic paralysis would involve an extended consideration of the development of the spinal cord, which would be out of place in a text book. The existence of anomalies in the cord, such as have been described by Ollivier, Longét, Goll, Calmeil, Charcot, Leyden and others<sup>1</sup> under the head of *Syringo-myelia* and *hydromelia* will explain development of early disease of the lateral columns. Leyden<sup>2</sup> has minutely described the openings found especially in the posterior columns as the result of myelitis.

In Leyden's cases the cavities which were the result of disease during foetal life were characterized by great unevenness of contour, by splitting up of the opening into others, or by certain indefinite and irregular variations, while the canals due to the absence of tissue incident to arrest of development were of symmetrical configuration; and the cord rarely presented any evidence of general disease, such for instance as sclerosis.

As to the arrest of development of the cord and the consequent abnormality in the lateral column function, we must take into account the fact of the existence of the transverse fissure alluded to by Charcot and others, among them Waldeyer. It is probable that the infantile forms of lateral column disease therefore are due either to some imperfect closure of the lateral column or a sclerosis beginning during uterine life.

Fig. 35.



(LEYDEN.)

1. Syringomyelia. 2. 3. Hydromyelia. a. a. Lateral fissures and imperfect development of lateral columns.

Flechsig,<sup>3</sup> in an elaborate article, has written extensively upon the connection of certain fibers in the lateral columns, with cells in the anterior

<sup>1</sup> See Prize Essay of American Medical Association upon Primary and Secondary degeneration of the Lateral Columns of the Spinal Cord, 1879, by the author.

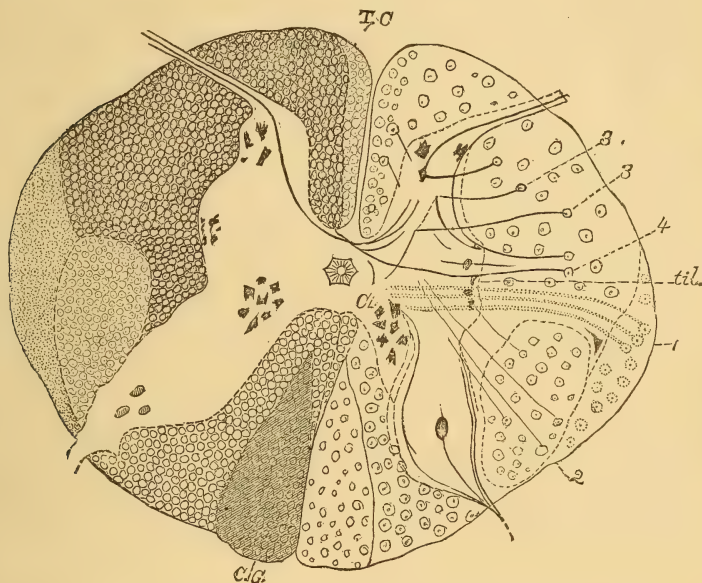
<sup>2</sup> Virchow's Archives, Bd. 68, Oct. 9, 1876.

<sup>3</sup> Archiv. der Heilkunde, 1877-1878.



gray horns and Clarke's columns and certain fibers of the crossed pyramidal columns evidently arise from large cells in the anterior parts of the anterior horns, and these are supposed by him to be concerned in the provision of peripheral motor power, and to be involved when there are contractures. Fig. 56 will enable the reader more fully to study his arrangement.

(Fig. 56.)



(FLECHSIG.)

T. C. Column of Türk. C. G. Column of Goll. C. L. Clarke's column. 1. Fibers in cerebellar column connecting with Clarke's column. 2. Connection of crossed pyramidal fibers with gray matter. 3. Connection of fibers of anterior column with cells of anterior cornua.

Gray<sup>1</sup> says that he is not prepared to accept Flechsig's views in their entirety, because he believes that disease of the crossed pyramidal columns is not always associated with contractures, and brings forward a case reported by Shaw in refutation in which the morbid processes involved the crossed pyramidal columns, and still this symptom did not occur.

The numerous cases of secondary degeneration after cerebral disease, in which contractures of the most formidable and conspicuous kind were manifested, and in which all degrees of degeneration, partial and complete were observed, would, however, rather neutralize the value of a single exceptional case; and such have been frequently reported.

The experiments of Woroschiloff<sup>2</sup> on animals have shown that the lateral columns of the cord contain motor and sensory fibers, which are

<sup>1</sup> Transactions of Kings Co. Medical Society.

<sup>2</sup> Ludwig's Arbeiten, 1875. Abstract in Journal of N. and M. Diseases.

variously distributed, and for the anterior part of the body the action of the latter is crossed, this action being more perfect in the fibers of the middle third of the lateral columns. There are also motor fibers in this part of the cord. His experiments show that irritation of the peripheral sensory nerves of the limb of an animal in front of the lesion of the cord, produces only reflex movements in the limb on one side, which is wholly or in part uninjured. If, however, this part of the lateral column is destroyed, it is impossible to cause reflex movements in the hind limb, even when excitation of the anterior part of the body is severe. It was found that if the anterior half of the lateral columns was not intact, no reflex movements could possibly be induced. Electrical excitation of the cervical cord "caused repeated alternate flexion or extension, or tetanic contraction of the limbs." The first would not follow if the middle third of the lateral columns was not intact. The clonic contractions took place even when the corresponding lateral column was destroyed. In regard to the production of the tendon-reflex, Schultz<sup>1</sup> and Furerbringer have experimented by dividing the cords of rabbits and exposing the tendons. They have come to the conclusion that the phenomena of tendon reflex are not those which result from a local excitation through muscles, nor that such movements are skin reflexes, but that there is local irritation of certain nerves described by Sachs,<sup>2</sup> which have terminal filaments in the tendons. We are also reminded by Erb,<sup>3</sup> that the tendon reflex occurs even when the tendon is tapped in situations where there is underlying bone, and where there is no possibility of jar or mechanical irritation of the attached muscles. The tendon in a relaxed condition can even be pinched when held in the fingers, and contraction will follow.

To do away with the possibility of cutaneous irritation, the skin may be anæsthetized by the local spray, and the same thing then occurs.

In some of Erb's cases, the tendon-reflex could be excited by pressure over one of the lumbar vertebræ, or over other bony prominences; but in this case there was no secondary reflex. In examples where irritation of the skin gives rise to the tendinous movement, the same are likewise secondary.

It has also been found that pressure on the central nerve will diminish, if not stop, the various expressions of heightened reflex in the lower extremities. The different phenomena of the tendinous reflex depend upon the integrity not only of sensory nerves, but the paths of sensory condition in the posterior columns; and Henz<sup>4</sup> observes that in certain hemiplegiæ connected with hemi-anæsthesia, the probable failure in producing tendinous and other reflexes depends not so much upon the paresis of the muscle, as upon the insensibility of the integument, or the nerves of

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<sup>1</sup> Centralblatt f. d. Med. Wiss., No. 54, 1875.

<sup>2</sup> Reichert and Du Bois Réymond's Archiv. iv., 1875, p. 402.

<sup>3</sup> Ziemssen's Cyclop., vol. xiii., p. 49.

<sup>4</sup> St. Petersburg Med. Wochenschrift, No. 35, Oct. 30, 1876.

the sinews. The central conditions which enter into the production of exaggerated states of the tendinous reflex, and of the reflex disorders of motility, are—

1st. A condition of irritation or inflammation of the central gray substance;

2nd. A suspension of inhibition.<sup>1</sup>

In this case the lesions involve the strands of nervous conducting matter concerned in the transmission of cerebral or local inhibition.

In the diseases under discussion, it would appear that the last of these is that which enters into the pathology of diseases of the lateral columns; for in the majority of cases the gray matter is intact.

The careful investigations of Flechsig, already referred to, demonstrate that certain fibers in the lateral columns are connected with certain cells in the anterior cornua and other parts; and that in disease affecting this part of the cord, the spinal inhibitory action which is acknowledged by nearly all neuro-physiologists, among them Erb,<sup>2</sup> Brunton,<sup>3</sup> and others, to enter into the production of certain motor impulses of spinal origin, is suspended.

Allusion has been made, in speaking of symptomatology, to the fact that an original excitation of a tendon was often followed by a series of muscular contractions.

This has been noticed by Freusberg; and according to Pfleuger<sup>4</sup> it is explained by the theory that the original excitation is transformed from sensory to a motor fiber on the same side of the cord; and then by others on the other side; thence back, following a zigzag course and giving rise to unequal muscular motorial innervation, and this will also explain the occurrence of transmitted reflexes to the other side of the body.

The contractions which occur are due to a tonic rigidity of the flexors and are rarely if ever attended by any change of substance or tissue of the muscle, but are due to an irritation of central nervous tracts.

The spastic gait is the result of reflex contraction of the muscles, dependent upon retractions of the tendons; and with this a certain paresis. The early sensory disturbances are due probably to irritation of the posterior nerve-roots, or perhaps to parts of the lateral columns which have been found by Ludwig and others to be concerned in the transmission of sensory impressions; and, as a consequence, the dragging neuralgic pains and burning.

<sup>1</sup> In regard to the suspension of cerebral inhibition, I think we may make use of the hysterical cases of lateral column degeneration to explain how an inveterate voluntary paralysis, such as occurs in hysteria, may by a continuous arrest of inhibition of the central variety, lead to a degeneration of parts concerned in the transmission of voluntary impressions.

<sup>2</sup> *Op. cit.*

<sup>3</sup> *West Riding Reports*, vol. iv.

<sup>4</sup> Quoted by Erb.



The gross appearances of disease of the lateral columns present many variations; and markedly differ in regard to situation and degree of degeneration. It is unusual to find absolute non-involvement of the other columns of the cord, as in the case observed by Westphal, and alluded to by Erb. The posterior columns are liable to be affected to some extent; and this complication affects very slightly the clinical features of this disease; while if there be involvement of the anterior columns, the conspicuous atrophy will give to the disease picture a very different aspect. This condition of affairs was witnessed in a case of anomalous progressive atrophy brought forward recently by Shaw,<sup>1</sup> and there are additional cases of this character reported by French authors.

So far, no autopsies have been made which revealed uncomplicated disease.

In a case which has been diagnosed by Charcot<sup>2</sup> to be one of pure "Tabes dorsalis spasmodique," the disease of the cord came more properly under the head of disseminated sclerosis than local degeneration. This case is mentioned in Bétou's thesis. Ollivier gives autopsical results, but these are too indefinitely detailed, and too inexact to be of much service. The cases, however, which are of greatest interest to us, are those in which there has been secondary disease. It has been assumed by Charcot, and in some of his hysterical cases it has been found, that the form of degeneration known as "primary" occupies a wedge-shaped area beginning at the cord, and extending through both the cerebellar and crossed pyramidal columns.

In one of his cases<sup>3</sup> the sclerosis was found to involve the entire length of both lateral columns, while other parts were perfectly healthy. There was no trace of meningitis, and the character of the semi-gelatinous, grayish change, was unmistakably sclerosis. The microscope revealed atrophy and disappearance of nerve tubes with annular constrictions. The gray matter was intact and the cells unaffected. There was increase of connective tissue and an abundant deposit of amyloid cells. In the descending secondary degeneration consecutive to cerebral disease, the lesion will be found on one side only, and the crossed pyramidal fibres will be affected; while if this descending form be seen as the result of spinal disease, the lesion will be bi-lateral and may involve other parts as well in the lateral columns at a different place.

An ascending lesion, according to Erb,<sup>4</sup> Pitres,<sup>5</sup> and others, is usually characterized by degeneration of a narrow peripheral border of tissue confined to the cortex and extending forwards somewhat as far as the anterior nerve-root tracts.

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<sup>1</sup> Journal of Mental and Nervous Diseases, January, 1879.

<sup>2</sup> *Léçons*, etc., 1878, p. 294.

<sup>3</sup> *Gaz. Hebdom.*, No. 7, 1865, p. 109.

<sup>4</sup> *Op. cit.* vol. xiii., p. 773.

<sup>5</sup> *Gaz. Med. de Paris*, 1877.

I have already sufficiently alluded to anomalies in development of the cord and the destruction of certain parts by disease before birth. Under this class comes the case detailed by Schultze,<sup>1</sup> in which, with hydrocephalus, there was congenital non-development of the spinal motor tracts and myelitis. Should the degeneration follow Pott's disease, Leyden<sup>2</sup> is of the opinion that it begins at the point of compression and extends downwards, although Michaud has found in some cases of slow compression that myelitis ascends in these columns. Should the cortex be involved primarily, and be the seat of a myelitis, it will be found that there is thickening of the neuroglia, from the periphery to the centre, just as in the primary sclerosis.

According to Lange, softening is a common form of degeneration, and the fibres of connective tissue are not uniformly thickened, but such increase of volume is detected here and there in the midst of the diseased mass, and irregularly-shaped nuclei will be found attached to their sides. In a case of my own, that of a girl who had died after suffering for some years from chronic myelitis (her limbs being contracted, especially the upper), it was found that the cord, especially in the cervical region, presented evidences of lateral sclerosis, which were more marked on the right side. A transverse section of the cord at the cervical region, under a low power, presented the appearances depicted in Fig. 57.<sup>3</sup>

Microscopic examination revealed on both sides a hyperplasia of connective tissue, which was most dense at the periphery of the cord, while there was a compact network of fibres which interlaced and extended to the centre. While the thickening was perceptible in the anterior root-zone, it was especially marked in the posterior part of the lateral columns. With a low power, a dark triangular segment of dense connective tissue was observed extending from the periphery to the outer border of the central gray matter. Extending posteriorly to a point limited by an imaginary line drawn from the posterior group of ganglion cells in the tractus intermedio lateralis to the border of the cord internally (*i. e.* adjacent to the gray matter of the posterior horns), was found a reticulated arrangement of thickened fibres, the interspaces becoming smaller, and the neuroglia more dense, until within a short distance of the direct fibres of the cerebellar column. At this part the spaces become larger and elongated, and the fibers more prominent. In the anterior part of this dense tissue were found arterioles with thickened walls surrounded by granular substance which had been thrown out. In the spaces between the thickened connective tissue, there was a general disappearance of nerve tubes, which was most conspicuous towards the periphery, where in certain localities but two or three fibers could be detected. The axis cylinders in some places were swollen, and there was a scattered deposit of granular substances, which was the result of "breaking down" of the connective tissue

<sup>1</sup> Centralblatt, No. 10, 1876.

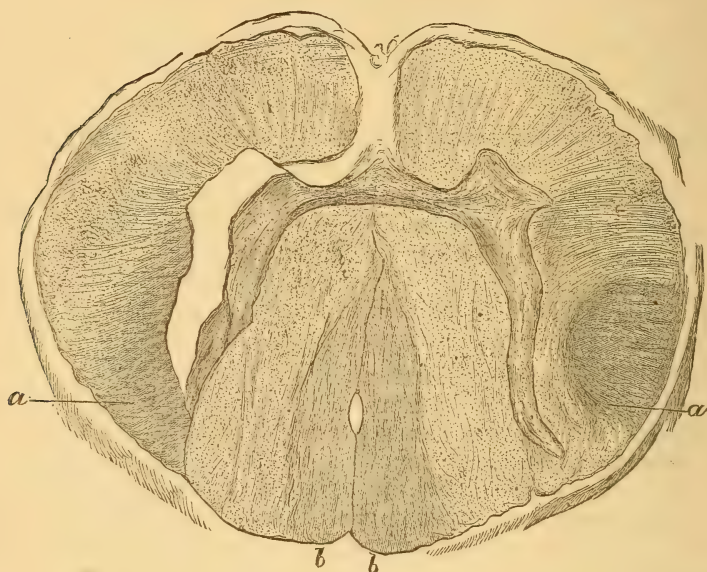
<sup>2</sup> Klinik der Rückenmarks krankheiten, Erste Band.

<sup>3</sup> Abst. in Schmidt's Jahrsbericht, 168, 1875, p. 238.

cells. The ganglion cells of both anterior horns were seemingly unaffected and their nuclei were distinct. The columns of Goll were also found to be the seat of sclerosis.

It is possible that in this affection there may be several grades of pathological trouble.

Fig. 57.



a. a. b. b. Sclerosed Tracts.

In Lange's<sup>1</sup> communication attention is directed to two conditions dependent upon varying degrees of diseased action:—1. Simple gray coloration without any destruction of nerve tubes. With this there is a prominence of neuroglia cells, while there is an increased clearness of the nuclei. The general discoloration is darker than in the next form. 2. Sclerosis, in which the main element is increase of connective tissue, and destruction of nerve tubes.

It is probable that these two conditions are those which are to be found in cases of slow progress, while a myelitis with softening is probably not uncommon in the secondary forms.

The changes which begin in simple discoloration and extend to sclerosis, include a list of slow changes of a progressive character. The nerve tubes appear at first altered in calibre and become swollen; their axis cylinders also swell, and there is unequal bulging of the membranes, giving rise to an appearance of varicosity. Granular degeneration is proba-

<sup>1</sup> Op. cit.



bly the next step in the process; and ultimately there is shrinking of the nerve fibers and disappearance. In secondary cases the existence of myelitis in other parts is to be observed; but the main appearance of the morbid process is to be localized. In many of these examples it is probable that the disease began by a simple ischæmic state, in some such pathological condition as the gray discoloration of Lange.

**Diagnosis.**—The diagnosis of disease of the lateral columns of the spinal cord is usually unattended by many difficulties; and the group of symptoms is too conspicuous and well-marked to permit the observer to err. Loss of power without atrophy, and reflex excitability without diminished sensibility enter into the formation of a unique train of symptoms; and unlike those of many spinal diseases, they are never separated in fully developed disease of this part of the cord. In speaking of the infantile form, I have alluded to several paralytic disorders of infancy with which it might be confounded. I have been fortunate enough to see a case of double talipes, the result of infantile paralysis, which at first suggested the disease in question; but even in this case the loss of electric muscular contractility (though there was not the extensive atrophy which might have been expected), led me to make a diagnosis of that much more common form of infantile disorder—infantile paralysis.

As to the primary disease of adult life, not much is to be said. It might possibly be mistaken for *transverse myelitis*, in which the urinary and vesical functions are involved, with bed-sores and anæsthesia. *Locomotor ataxia* cannot be mistaken for the disease; for in certainly half if not more of the cases the patellar tendon-reflex is absent. There are, in addition, the symptoms of ataxia, loss of muscular sense, anæsthesia of the tactile variety, optic nerve disease, and visceral pains.

The gait in the two diseases is radically different. In *locomotor ataxia*, the patient throws out his feet, coming down on his heels; while in all forms of *degeneration of the lateral columns*, as has been shown, there is a tendency to walk on the toes—the feet seem to cling to the ground; and there is adduction of the thighs.

In *disseminated sclerosis*, there is usually tremor, irregular involvement of the extremities, cephalic disorders, and generally more or less ataxia.

*Chronic myelitis of the anterior columns*, *progressive muscular atrophy*, and *amyotrophic lateral sclerosis*, may all resemble, in certain features, the disease under consideration—although if atrophy is conspicuous, the diagnosis is easy enough; but occasionally, an anomalous case is sufficiently puzzling to create a doubt.

I have seen a case of progressive muscular atrophy of the lower extremities which presented exaggerated reflex excitement of the tendons. There was a general trepidation which could not possibly be mistaken for the unequal muscular contractions known as *vermicular tremor*; but in this case there was a difference of an inch and a half in the circumference of the thigh, and the muscles of the neck were unevenly atrophied;

while added to the features of progressive muscular atrophy there was a commencing aphonia and other bulbar symptoms.

In *adult chronic spinal paralysis* the development of the disease may be so insidious as to give rise to a reasonable suspicion as to its true nature; and should there be added thereto slow contractions of the dorsal muscles, the diagnosis will be still more puzzling. In such cases the tendon-reflex will be found to be lowered or absent, and the contractures which result are not those of the spastic variety, but rather of paralyzed muscles opposed to those which are not.

The forms of secondary origin are more difficult to recognize; for complicating symptoms play a part which greatly confuses the observer. There may be all the symptoms of unequal congestion, of myelitis, or of concussion of parts other than the lateral columns; and time is required before it is possible to arrive at a conclusion.

In such cases, the involvement of parts above the lesion is significant. A paraplegia of the lower extremities may exist and be connected with anæsthesia, retention and incontinence, and even bed-sores; but at a later period, the arms may become the seat of paresis without anæsthesia, but with highly-developed tendon-reflex; and at this stage it is possible to find diminution of the symptoms of an inferior transverse myelitis, the anæsthesia clearing away, the function of the bowels and bladder being restored, and the gait becoming changed; while to the paresis there is added an excitement of the tendon-reflex; but of course such cases are rare. Should there be anæsthesia, however, the case may be supposed to be one of central or posterior myelitis. The diagnosis of hysteria is sometimes attended with difficulty; but it may be borne in mind that the paralysis is one of a purely *voluntary* kind in the beginning.

In this connection the reader is referred to an admirable article by Ferber and Gasser<sup>1</sup> upon certain forms of contractures of the hands and fingers who report the case of E. Gull, a woman 39 years old, who had suffered for some time from irregular menstruation; and for several years there had been periodic contractions of the hand and fingers. Afterwards the hands became permanently contracted, there being spasms of the flexors without any atrophy whatever. The muscles contracted were the common flexors of the hands, as well as the adductors of the thumbs and interossei. Dr. Buzzard in a recent communication to the *Lancet*<sup>2</sup> speaks of the difficulty of diagnosing the cases of hysterical origin from those of a purely organic character.

**Prognosis.**—Infantile cases may be said to be utterly hopeless except when secondary to Pott's disease; and neither medical or surgical treatment have so far proved of the least permanent benefit.

With cases of primary degeneration, or of the functional form, the matter is different. Erb has spoken of improvement; and the cases brought forward by Kussmaul, Berger and others, show that the progno-

<sup>1</sup> *Archiv. für Psychiatrie, etc.*, vij., p. 140, 1877.

London *Lancet*, June, 1881.

sis is not utterly bad, and Nixon's case of antero-lateral sclerosis was greatly benefited by the remedies of which I have spoken.

Hysterical cases are always rebellious, as the central disease is the consequence of a long existing nervous condition; and not only is the psychical influence difficult to combat, but the degeneration itself is of so extensive a character that it defies successful treatment. Strange to say, secondary affections of spinal origin are not utterly beyond the reach of treatment. This is especially true in secondary degeneration or local disturbance after concussion. Erichsen<sup>1</sup> in his well-known work alludes to certain cases in which there was quite extensive spinal trouble from railroad injury with symptoms indicative of lateral column disease, and yet recovery took place. Should such concussion be unattended by laceration of nervous substance, there is some chance for improvement.

Should there be extension upwards, as occurs sometimes in both the *primary* and secondary form of disease, there may be bulbar symptoms and death; while if the cervical region of the cord be the district ultimately attacked, serious pulmonary symptoms may be added to those of the disease.

**Treatment:** In the favorable case treated by Kussmaul, the chloride of gold and sodium in doses of gr.  $\frac{1}{3}$  t. i. d., was used until the patient had taken ninety grains in all. Erb places nitrate of silver at the head of the list of drugs, and at the same time recommends hydropathy.

It would seem that Nixon<sup>2</sup> also has found benefit to follow the use of the silver salt; and much improvement followed in his case under the continued administration of the following prescription:

R  
 Argenti Nitratis,  
 Ext. Nucis Vomicae, ʒʒ gr. jv.  
 Ext. Gentianae, q. s.  
 ℞. Divid in pill No. xii.  
 Sig. One ter in die.

Charcot is also in favor of the nitrate of silver, and suggests in addition the application of the cautery along the spine. He has used the bromide of ammonium and sodium in large doses to diminish trepidation and spasms, and has met with excellent results.

Paradism by means of the wire brush is recommended, and galvanism (the continuous current) has been of service in Erb's hands. Strychnine is advised against when there is so much reflex disturbance. Thermal saline spring baths have been praised; but it has been found that internally the waters do no good.

In my own practice, I have found that the Fl. Ext. of Conium in doses of five minims thrice daily to be increased, is the best remedy to diminish the violence of the trepidation, while belladonna or the sulph.

<sup>1</sup> Concussion of the spine and nervous shock, etc.

<sup>2</sup> Dublin Monthly Journal, vol. lviii., 1874, p. 207.



of atropia in local hypodermic injections of grs.  $\frac{1}{8}$ , is useful when there is great spastic rigidity. Hyoseyamia acts well and is an admirable anti-spasmodic.

As to other internal remedies, I have given phosphorus, the nitrate of silver, and chloride of gold a fair trial, but am disposed to place more reliance on cod-liver oil, ergot, or some salt of mercury for the treatment of the central disease, fully believing that nutrition should be improved and the local circulation modified.

I have found that the utmost quiet is necessary not only for the comfort of the patient, but for the amelioration of the disease. He should be kept still, and not allowed to take fatiguing exercise; and all sources of reflex excitation should be avoided. For the hysterical cases, we should pursue a different course and endeavor to make them bring into use the muscles of the affected limb. For cases of secondary origin, the galvanic current seems to possess great advantages; and should there be meningeal troubles, the administration of ergot is to be pushed. If the case be like one reported by Leyden, and probably rightly supposed by Erb to be of specific origin, it is necessary to give the iodide of potassium.

### TETANUS.

**Synonyms.**—Rigor nervosus; Mal de cerf; Tétanos (Fr.); Locked jaw.

**Definition.**—Tetanus is an affection characterized by tonic spasms of a great number of muscles, particularly those of the jaw, neck, back, and lower extremities. It is never attended by loss of consciousness, and nearly always approaches an unfavorable termination. It is a disease which may be either idiopathic or traumatic, and is not confined to any age or sex, as it may be a condition at birth (trismus nascentium), or occur at any subsequent time.

**Symptoms.**—The more familiar examples follow wounds, and such injuries may be exceedingly slight—the puncture of a rusty nail, a needle or a blunt instrument being often likely to give rise to the attack; or it may be of distinctly idiopathic origin. The first symptoms generally noticed are a stiffness of the neck, a slight soreness of the throat, and a contraction of the jaws so that it may be difficult for the patient to open his mouth. There may be general malaise and discomfort, which may last for several days, and the patient is unable to masticate or swallow his food properly, and consequently eats but little. He may think that he has simply caught cold, and neglect to seek medical advice; but new developments will show the condition to be more serious than he imagines.

The closure of the jaw may become more complete, and within the next twenty-four hours (the fourth or fifth day of the affection) he will show unmistakable signs of the increasing violence of the disease. His face wears the peculiar expression which has been called the *risus sardonicus*, the features appearing pinched and set, and the corners of the mouth are

drawn upwards, while the eyes are prominent and the hair and eyebrows quite bristling. The brows are knit, and there is a characteristic appearance, which, if once seen, cannot be mistaken. Radcliffe considers the *risus sardonius* quite pathognomonic of tetanus. Pain in the epigastrium becomes very severe, and is not relieved by medicine. It is impossible sometimes to open the jaws even when we desire to give food or medicine, and it is sometimes necessary to use quills and other delicate tubes for the purpose of feeding. Spasms of the pharyngeal muscles may also defeat all attempts of this kind, for, even if the teeth are parted and nourishment is inserted, the food is forced with great violence through the nostrils. Other spasms now mark the progress of the disease. The muscles of the back begin to be convulsed, and finally those of the lower extremities, and as a consequence we observe the appearance of *opisthotonos*, which is an extremely striking symptom, and much more common than *emprosthotonos*, which may also take place, or *pleurosthotonos*. It is hardly necessary to say that *opisthotonos* is the result of a tonic spasm of the muscles of the back, so that the patient's body describes an arc, the head and heels touching the surface upon which he is lying, and the middle of the back being raised some distance therefrom. When the body is bent in the opposite direction—forward—the condition is known as *emprosthotonos*; and when the muscles upon one side of the body are contracted we designate the lateral curve produced as *pleurosthotonos*. During this tonic convulsive state individual muscles may be the seat of painful spasms, which are very agonizing. Muscles have been torn across and bones broken by the great strain, and the force exerted is something wonderful. The tongue is rarely affected, and the hands are not usually at any time rigid or contracted. The spasms are easily produced by slight agencies, as reflex irritability is decidedly exaggerated. Jarring the bed, tickling of the soles, or a draught of air allowed to blow upon the surface will immediately bring them on. This convulsive stage lasts until death, but when the end is approaching becomes less sthenic as the patient grows more and more exhausted. There may be an occasional severe paroxysm before death, but it is not at all like the form of violent convulsion of the middle stages. The pulse throughout the developed disease is very rapid and fluttering and ranges between 120 and 140, and the respiratory movements are irregular and catching, as the spasms affect the muscles of the thorax as well as others which are directly concerned in this process. Dyspnoea is very distressing, and is expressed between the seizures by much gasping and anxiety of countenance. The skin is dark, and large rings about the eyes are indicative of collapse, while the face of the victim is haggard and depressed. The patient perspires quite profusely, and the skin is excessively hot; and a prominent feature of tetanus is the marked elevation of temperature, which rises even sometimes as high as  $110^{\circ}$ , and actually reaches a higher point after death. In a case observed by Wunderlich<sup>1</sup> there was a marvellous elevation of this kind, and a very tardy fall after death.

<sup>1</sup> Archiv. der Heilkunde, Bd. ii., and v. (1861–63). Reported by Radcliffe.

| Date.                                    | Respiration. | Pulse. | Temperature<br>(Fahrenheit). |
|--|--------------|--------|------------------------------|
| 24th July, 1861 . . . .                  | 24           | 96     | 102°                         |
| 25th " " . . . .                         | 22           | 82     | 102                          |
| 26th " " 9 A. M. . . .                   | 20           | 96     | 104.45                       |
| " " " 6 P. M. . . .                      | 32           | 112    | 103.55                       |
| " " " 9.20 P. M. . . .                   | 36           | 180    | 110.1                        |
| " " " 9.35 P. M., <i>death</i> , . . . . | . . .        | . . .  | 112.55                       |
| after death, 2' . . . .                  | . . .        | . . .  | 112.77                       |
| " " 5' . . . .                           | . . .        | . . .  | 113                          |
| " " 20' . . . .                          | . . .        | . . .  | 113.22                       |
| " " 35' . . . .                          | . . .        | . . .  | 113.55                       |
| " " 55' . . . .                          | . . .        | . . .  | 113.67                       |
| " " 60' . . . .                          | . . .        | . . .  | 113.55                       |
| " " 70' . . . .                          | . . .        | . . .  | 113.22                       |
| " " 90' . . . .                          | . . .        | . . .  | 113                          |
| " " 100' . . . .                         | . . .        | . . .  | 111.8                        |
| " " 6 hours . . . .                      | . . .        | . . .  | 106.25                       |
| " " 9 " . . . .                          | . . .        | . . .  | 104                          |
| " " 12 " . . . .                         | . . .        | . . .  | 102                          |
| " " 13½ " . . . .                        | . . .        | . . .  | 101                          |

Dr. Joseph Jones, of New Orleans, the author of one of the most able articles upon this subject that has ever appeared, has made numerous examinations of the urine. He found that the quantity of urine excreted during the "active stages was greatly diminished from the normal standard, and in the successful cases treated the amount increased with subsidence of the symptoms." He also found that the urea was increased during the active stages, and the uric acid was diminished.

The diminution of the excretion of urine is by him supposed to be accounted for by the small quantity of fluids taken, and by the loss of liquid in profuse perspiration.

The mind is perfectly clear throughout the disease, and the patient suffers great mental misery as he fully realizes his terrible condition; and sleep is nearly always absent, this being one of the most distressing features of the disease. If this is obtained, even in brief snatches, the muscles are relaxed, and all spasms disappear for the time, but immediately reappear upon awaking. The probable cause of death is either the closure of the glottis, or exhaustion, which is an inevitable result of the violent muscular action. In new-born children the disease sometimes appears between the first and fifth days, the first symptoms noted being restlessness, trembling of the lower jaw, and desire for the breast, which



the child leaves almost immediately. At the end of twenty-four hours, or even earlier, the muscles of the jaw are felt to be contracted and rigid, and it cannot open its mouth; there is a peculiarly aged expression upon its face, the skin of the forehead being wrinkled. The eyelids are closed, and the lips are compressed over the teeth. The head is drawn back, and general spasms of the muscles of the back follow. Periods of remission occur, and the patient is thrown into a paroxysm by the most trivial agencies. The skin is very red and dark, and after a series of paroxysms, which may continue for several days, death closes the scene.

**Causes.**—Exposure to damp and cold are the only known exciting causes of the idiopathic variety; and traumatisms of certain kinds, or accidents during parturition, precede the other form. A punctured wound, which may be received from a nail or splinter, is much more likely to give rise to tetanus than an incised wound; and injuries in which there is mangling or crushing of muscular tissue are frequently concerned in the production of the disease. Railroad injuries are therefore especially dangerous. Tetanus sometimes follows surgical operations, and it has been thought in these cases to depend upon partial section of some nerve-trunk. Dupuytren<sup>1</sup> goes far enough to recommend re-amputation. It may be stated that in certain regions there are apparent endemic influences at the time of such predisposition, when any surgical operation may have this termination. This local influence prevails in Cuba and other tropical countries, and in Long Island and in other parts of the American seaboard.

Jones has collected the statistics of tetanus, and the following table shows its prevalence in hot climates:—

| Place.         | Period.   | Total deaths. | Deaths from tetanus. | Proportion. |
|----------------|-----------|---------------|----------------------|-------------|
| London . . .   | 1850-3-4  | 224,515       | 73                   | 1 in 3075   |
| Ireland . . .  | 1831-1851 | 1,187,374     | 238                  | 1 in 4987   |
| New York . . . | 1819-1834 | 83,783        | 112                  | 1 in 748    |
| Bombay . . .   | 1851-1853 | 42,651        | 912                  | 1 in 46     |

I am indebted to Dr. Charles Findlay, of Havana, Cuba, for the following concise table, which shows the prevalence of the disease in that island:—

<sup>1</sup> *Léçons Orales*, tome ii. pp. 599-612.

|            | 1872.   |          | 1873.   |          | 1874.   |          | 1875.   |          | 1876.   |          | Average. |          |   |
|------------|---------|----------|---------|----------|---------|----------|---------|----------|---------|----------|----------|----------|---|
|            | Adults. | Infants. | Adults. | Infants. | Adults. | Infants. | Adults. | Infants. | Adults. | Infants. | Adults.  | Infants. |   |
| January,   | 4       | 47       | 4       | 39       | 3       | 34       | 4       | 33       | 6       | 17       | 4.2      | 34.0     | Pop. of Havana,<br>250,000.                           |
| February,  | 5       | 29       | 1       | 30       | 3       | 18       | 4       | 30       | 4       | 30       | 3.4      | 27.4     | Births per annum,<br>5000.                            |
| March,     | 6       | 24       | 3       | 28       | 4       | 31       | 5       | 24       | 4       | 29       | 4.4      | 27.2     | Deaths by tetanus in                                  |
| April,     | 6       | 26       | 5       | 30       | 0       | 24       | 4       | 18       | 5       | 26       | 4.0      | 24.8     | Adults=0.192 a year<br>per 1000 inhabit'rs.           |
| May,       | 3       | 27       | 1       | 29       | 3       | 33       | 5       | 30       | 3       | 35       | 3.0      | 30.8     | Death of infantile<br>tetanus.<br>per hun' red births |
| June,      | 2       | 24       | 3       | 33       | 2       | 36       | 5       | 29       | 5       | 39       | 3.4      | 32.2     |   |
| July,      | 4       | 25       | 5       | 20       | 4       | 31       | 3       | 36       | 3       | 35       | 3.8      | 29.4     | 7½  |
| August,    | 3       | 35       | 5       | 33       | 5       | 45       | 5       | 38       | 2       | 46       | 4.0      | 37.4     |   |
| September, | 3       | 28       | 1       | 29       | 3       | 41       | 3       | 42       | 6       | 33       | 3.2      | 34.6     |   |
| October,   | 1       | 42       | 6       | 32       | 3       | 36       | 1       | 43       | 4       | 37       | 3.0      | 38.0     |   |
| November,  | 6       | 45       | 4       | 42       | 4       | 29       | 3       | 37       | 6       | 41       | 4.6      | 38.8     |   |
| December,  | 2       | 36       | 4       | 23       | 4       | 31       | 5       | 28       | 7       | 40       | 4.3      | 31.6     |   |
| 12 months, | 45      | 388      | 42      | 368      | 38      | 389      | 47      | 388      | 55      | 408      | 48.4     | 382.2    | Yearly average.                                       |
|            |         |          |         |          |         |          |         |          |         |          | 4.0      | 31.8     | Monthly average.                                      |

Long Island, it seems, has gained an unenviable notoriety as a place where tetanus is exceedingly common; but it will be seen that there is much exaggeration in the reports which, as a rule, come to us in the newspapers, and which are nearly always sensational. I have devoted some time to the investigation of the subject, and have written to several well known physicians of eastern Long Island, and have received two or three letters in reply.

Dr. Stilwell, an old settler of Sag Harbor, whose opportunities for research have been quite extensive, writes as follows: "About 20 years ago I came to this place to practice, and learning the fact of the prevalence of tetanus, or its liability from certain accidents, I attempted an investigation, but failed of any success or satisfaction. Several supposed cases having recovered naturally brought many cases under my observation, but most of them died. Several did not, and from my after-remarks here you will perceive the reason. I have never known the disease to exist as an epidemic, but it is apt at certain seasons of the year, to follow wounds. Hot and damp weather, with cool evenings, is its favorite season." The Doctor has known but two instances of recovery from traumatic tetanus.

When a patient has recovered from tetanus it has been by a very slow process, the period between the spasms lengthening until they finally disappeared. Under favorable circumstances this required several weeks. "I have known fatal cases of idiopathic tetanus in July and August

caused by fatigue and overheating, and sitting down to cool off in the ocean breezes. Farmers have often informed me that the *white frost* on grass would give cattle lockjaw. I have known a horse driven to fatigue turned out to pasture in a cool night when white frost formed upon the grass, and die with tetanus. I have known horses, in the heat of summer driven seven miles to the seashore and there cooled off in the ocean breezes, die of the same disease. The multiplicity of cases occur in summer and in the heated term with cool nights. A farmer bruised his thumb-nail and pulled turnips in a frosted field; he died of tetanus." The other letters I have received are in substance very much like that of Dr. Stilwell, and none of them suggest that the disease is as frequent as it is generally supposed to be. Dr. Benjamin, of Riverhead, says: "I have practised thirty years in this village, have an average of about one case each year (others claim twice that number), and should think the other physicians in the Assembly District would average about the same; if so, it would make nineteen cases each year with a population of 19,000. My opinion is that there has been no marked change in the past forty years as to its frequency or fatality. A very large proportion of our cases prove fatal in from one to three days. Of *trismus nascentium* I have had six cases during the past thirty years, all of which were fatal." The information that I have derived from popular sources is, however, somewhat contradictory. I learn that about Good Ground, which is nearly twenty miles west of Sag Harbor, there are times when traumatic tetanus is very common; and it is not safe for any person who has received even the most trivial injury to remain in the neighborhood.

Capt. Foster and Capt. Joseph Penny, of Ponquogue, which is upon the sea-coast, state that they have known of tetanus, which was very common at certain seasons; several of their friends have died, and others have moved temporarily from the place as soon as injured. It was not uncommon for women about to be confined to leave the locality; and cases of *trismus neonatorum* were of quite frequent occurrence. One man whose foot had been crushed by a horse died in a few days.

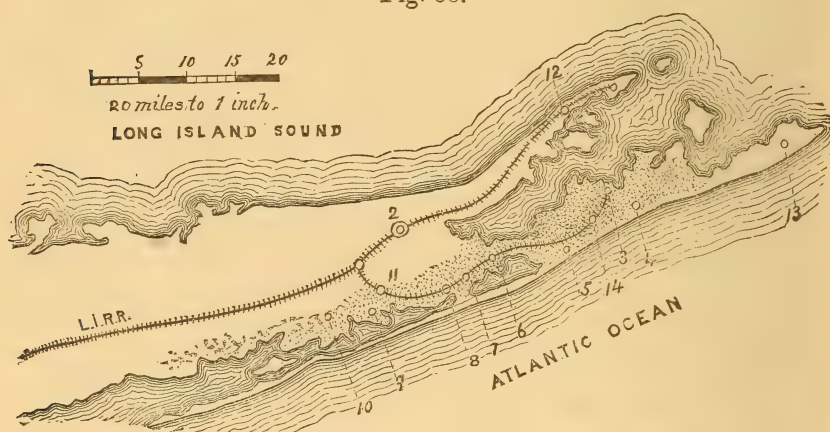
From Mr. Wells, of Quogue, I ascertained that the disease is confined almost entirely to the district extending from Moriches to East Hampton, and that at the extreme easterly end of the Island (Montauk Point) no case has been known to occur. So perfect is the immunity at this place, that colts are taken there to be castrated and not removed until the wound is healed. The disease is more common during the fall than at any other season. Mr. Wells has known of from twenty to twenty-five cases, mostly men and boys, in a district forty miles long, during the past five years. In this region castrated colts generally die soon after the operation. In one case, of which my informant knew, a man was shooting ducks in a battery; his shot-gun accidentally went off, the charge removing about one-half of the great toe. The wound was not especially painful, but at the end of eight days convulsions began, and he died in thirty-six hours.

Mr. White, of South Hampton, scratched his thumb with a briar in the



field, and afterwards died. Mr. Hand, of Canoe Place, died after a slight injury to the ankle. Mr. Wells also told me that several cases followed wounds received in the field where a form of shell-fish known as the "horse shoe" (king-crab) is used for manure. By the fall these crawfish have undergone advanced decomposition, and their long spines, which project in any direction, are very apt to wound the bare-footed field hand. These statements are entitled to some credence, for the doctor was very often not called in. At the eastern end of the island several cases of fatal tetanus within a very short time occurred in the practice of Dr. Trudeau, then of Little Neck. Along the Atlantic sea-board I am told that this disease is by no means uncommon, and that on the Southern sea-coast it is much more frequently met with than in higher latitudes. In a very interesting communication from Dr. Findlay, of Havana, he mentions a case in which the application of a blister in a case of pleurisy was followed by fatal tetanus. The accompanying map will enable the reader to perceive the geographical distribution of endemic tetanus on Long Island, the dark spots showing the limit of the region, and the points where it prevails to the greatest extent.

Fig. 58.



MAP OF SUFFOLK COUNTY, LONG ISLAND.—1. Manor. 2. Riverhead. 3. Sag Harbor. 4. East Hampton. 5. South Hampton. 6. Ponquogue and Good Ground. 7. Quogue. 8. West Hampton. 9. East Moriches. 10. Centre Moriches. 11. Seatuck. 12. Greenport. 13. Montauk Point. 14. Bridge Hampton. Darkest spots indicate points of greatest prevalence.

Cold climates have something to do with the production of tetanus, as we would infer from Dr Kane's statement that intense cold produced "an anomalous spasmodic affection allied to tetanus," which affected most of his party, destroyed two men, and killed all his dogs. Trismus neonatorum is supposed by Vogel<sup>1</sup> to depend upon the formation of the cicatrix when the umbilical cord is roughly handled, and there is probably pressure of some nerve by the contraction of the cicatrix.

<sup>1</sup> Diseases of Children, p. 65. Translation by Raphael, N. Y., 1870.

Frost-bite may sometimes give rise to tetanus, and the following cases are examples of this kind :

They occurred under the care of Dr. Bethune, of Toronto. The first was that of a farmer who was exposed to intense cold for about three hours while driving. His feet and fingers became severely frost-bitten without his becoming aware of the fact until he arrived home. On admission to the Toronto General Hospital, four days later, the toes and the greater part of both feet were found in a condition of moist gangrene.

The fingers and parts of both hands on the dorsal surface were black and dry. Four days after admission he was seized with tetanic symptoms, which rapidly developed. Chloral hydrate in thirty-grain doses, with extract of Calabar bean in one-fourth-grain hypodermic doses, until five grains had been given, failed to combat the disease, and the patient died in thirty hours after the accession of the attack.

The second case was that of a man who, having lain out in a barn all night, had both feet severely frost-bitten, subsequently becoming partially gangrenous. In this case trismus set in nine days after exposure, and soon developed into well-marked tetanus, to which the patient succumbed in about thirty hours.<sup>1</sup>

**Morbid Anatomy and Pathology.**—The older writers have written a great deal in regard to the morbid anatomy of tetanus; but the collected facts throw no light upon the pathology, and are to a great degree valueless.

Lockhart Clark<sup>2</sup> in 1865 found in six cases that there was degeneration of the gray substance of the cord. "The first case was reported at some length, and the lesion was found more or less from the origin of the second cervical nerves to the lumbar enlargement. At the second cervical nerve, streaks and irregular areas of disintegration were observed in different parts of the gray substance, and particularly around the central canal, on the right side of which was a space of considerable size containing a finely granular fluid, with the debris of blood-vessels and nerves. The posterior and lateral white columns, especially along the edge of the various fissures which transmit blood-vessels, were damaged in a similar way, and in some sections the deeper portions of the posterior columns which rest upon the transverse commissure were softened to a considerable degree. This disintegration was still more marked in the cervical enlargement, chiefly behind and at the sides of the canal. The posterior commissure was wholly and the anterior partially destroyed by a fluid transparent and granular area. Throughout the cervical enlargement similar lesions were discovered, varying from a state of softening to one of complete solution, and diminishing at intervals or almost disappearing, to return shortly in the same form. At the upper part of the dorsal region the shape of the cord was much altered, and extensive lesions of the same kind were everywhere seen. In both lateral halves of the gray

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<sup>1</sup> London Lancet, March, 1875.

<sup>2</sup> Med.-Chir. Trans., 1848 and 1865, and Med. Times and Gazette, 1865.

substance, the left lateral columns, the right antero-lateral column, the superficial portion of the anterior columns, and in the posterior columns similar appearances were found. Below this point there was less disease as far as the fourth dorsal vertebra. Here, in addition to the areas of disintegration, large extravasations of blood were found along the whole lateral part of the gray substance on both sides of some sections, in one side only of others; while the lumbar region manifested the same lesions as the cervical."

Dr. James Tyson<sup>1</sup> has detailed two cases in which softening of the posterior columns occurred. In one of these there was extravasation of blood in the posterior columns, and to some extent from the vessels of the pia mater. The central gray commissure was destroyed. In the other case no extravasation was found in the posterior columns, but there was venous congestion of the dura mater. I was presented by Prof. L. McLane Tiffany, of Baltimore, with a piece of the cord of one of his patients who had died with tetanus following a severe burn. The pia mater was greatly thickened, and the small posterior arteries were enlarged. Throughout the section, which was viewed at first with a low power objective, I perceived a rather extensive increase of the neuroglia. The anterior nerve-roots appeared to be very well defined. Throughout the white and gray matter there was visible numerous round cells quite translucent and bright, which resembled somewhat colloid bodies. These were more plentiful in the posterior column. The vessels of the gray matter were all more or less enlarged, and some of them were surrounded by spaces which were considerably wider than the diameter of the vessel. The cells of the anterior cornua were quite disintegrated, and some had taken an oval form. Those that could be recognized were found to have broken processes, and many had granular contents. The nerve-trunks were unaffected.

Arlong<sup>2</sup> and Tripier, Erichsen, and Bouillaud found that the end of the nerve in the wound was diseased, and Lepelletier<sup>3</sup> and Froriep<sup>4</sup> discovered in one case that the neurilemma of the nerves in the vicinity was the seat of inflammatory changes, which extended from the periphery to the cord. This latter appearance indicates an exceptional condition of affairs, and as for the nerve-change in the wound, it is not to be wondered at, for if there is any importance to be attached to the circumstance of the morbid appearance of an injured nerve, it is certainly inconsiderable when we consider how frequent must be such a pathological condition, and still there is not a proportionate amount of tetanus.

Dr. R. W. Amidon,<sup>5</sup> has lately published very full notes upon a case of tetanus, which throw some light upon the question of morbid anatomy. In this observation the disease followed an injury of the median nerve,

<sup>1</sup> The Practitioner, August, 1877.

<sup>2</sup> Archives de Physiol., etc., 1870.

<sup>3</sup> R vue M dicale, iv., 1827.

<sup>4</sup> Neue Notizen, 1837.

<sup>5</sup> Some new points on the Path. Anatomy of Tetanus, Archives of Medicine, June, 1879.



and the patient died five days afterwards. Microscopical examination revealed a variety of interesting meningeal, vascular and other lesions—those claiming our attention chiefly being the presence of vacuoles in the medulla and very decided changes in the region of the spinal accessory root-fibers especially, while the vagus, hypo-glossal and glosso-pharyngeal nerves were found to be the seat of vascular lesions. The symptoms pointing to implication of these nerves were quite pronounced.

Our knowledge of the pathology of tetanus is based almost entirely upon the experiments of physiologists, and we are left somewhat in the dark as to the questions: 1. Whether it is a central disease resulting from a morbid peripheral irritation which is reflected upon the cord. 2. Whether it is a central disease *per se*, and the appearances noted after death are primary. 3. Whether the morbid changes are secondary to the symptoms, and due to mechanical causes.

We have so far been taught how general spasm may be produced. Mitchell<sup>1</sup> and Morehouse caused in animals very violent convulsions by injecting into the vertebral canal a half ounce of fluid, and very hot or very cold water seemed to aggravate the spasms. Cold applied to the spine, whether produced by the rhigoline spray or by ice, gave rise to the same phenomena. Cold to the medulla caused the animal to topple backward.

Upon examination the vessels were found to be intensely congested. So far, we are furnished with the first link in our chain. Assuming that the spasmodic movements are due to a congestion of the cord, and conceding that pathological anatomy has furnished us in nearly every instance with evidence of congestion of the gray matter, we are to discover what is the factor of such congestion. It may depend upon a reflected impression transmitted to the vaso-dilators, or it may depend upon local irritation by impure blood which produces secondary hyperæmia. In strychnine poisoning, the symptoms of which resemble those of tetanus very closely, the spasmodic phenomena are undoubtedly due to the imperfect oxygenation of the blood; consequently the cord is supplied with blood loaded with carbonic oxide. It seems to me very possible that the same condition of affairs exists in tetanus; that there may be direct irritation of the nervous matter of the cord dependent upon some primary blood condition.

Fox<sup>2</sup> very clearly expresses himself as follows: "The abnormal blood imperfectly nourished the cord. An imperfectly nourished cord is *ipso facto* an excitable, an impressible cord; this impressibility renders arterial spasms abnormally facile, whether the exciting cause is the circulation in the cord of more of the morbid blood, or reflected irritation from a diseased nerve at the periphery, or reflex irritation from any other cause and from any other point in the body, and if this arterial contraction goes on for any protracted period, or is frequently repeated, we may

<sup>1</sup> Am. Journ. Med. Sciences, 1866.

<sup>2</sup> Op. cit., p. 362.

find various lesions due to imperfect blood-supply in addition to those due to diminished nutrition from the original nature of the blood, while, as a sequence of the spasmodic arterial contractions, we get hyperæmia, and perhaps exudation, and lastly the pressure of the exudation or some peculiarity in its nature may lead to some disintegration of the nervous centres."

This theory seems to me to be tenable for several reasons: 1. Injuries of peripheral nerves are common, and the cases of resulting tetanus are out of all proportion to those presenting no subsequent nervous symptoms. 2. Its endemic nature, its prevalence in certain districts, and its not uncommon idiopathic origin when there is no ascertained eccentric cause. 3. The appearances of the cord are of a destructive character, and it is a matter of doubt whether they are not more a result than a cause.

Considerable discussion has taken place in regard to the cause of the high elevation of temperature. Verneuil does not consider it due either to myelitis of the superior part of the cord, or to asphyxia or muscular contractions; but Mason is decidedly of the opinion that such increase in temperature is alone the result of muscular action. The experiment of Mason has shown that the temperature of a tetanized muscle is often increased from one to two degrees.

The medulla has been found to be the seat of grave lesions, such as in Amidon's case for example, and it is probable that the trismus and other evidences of an excited state of cranial nerve innervation, which occur in the beginning, are indications of primary disturbances in the bulb.

**Diagnosis.**—The diseases and conditions with which tetanus may be confounded are *hydrophobia*, *strychnine poisoning*, *hysteria*, and *acute spinal meningitis*. In the first there is no risus sardonicus; the convulsions are clonic; there is the noisy hawking and effort to spit; the dread of water, the delirium, and finally the history of a bite by a rabid animal, which, however, is not always ascertained. Strychnine poisoning is very easily mistaken for tetanus. In poisoning by a large dose of the alkaloid the symptoms appear rapidly, and death takes place in a short time. The hands are clenched and rigid, but the jaw can be opened, which is not possible in tetanus. This resemblance between the two conditions has been made use of in more than one poisoning case as a ground of defence, and in that of Cooke, who was poisoned by Palmer, the question was narrowed down to the appearance of the cord. Cases of hysteria sometimes present symptoms which not rarely counterfeit those of tetanus. The jaw may be locked, but there will be few of the other features. Hysterical patients are nearly always seemingly unconscious, and there are no evidences of suffering whatever. In spinal meningitis the muscular rigidity seems to be dependent, in a great measure, upon the patient's efforts to relieve the pain which is produced by an uncomfortable position. The locked jaw, which is an early symptom of tetanus, is absent in acute spinal meningitis.

**Prognosis.**—Dr. Jones<sup>1</sup> has collected 480 cases of tetanus, 213 of which recovered under treatment, the mortality being 49.2 per cent., or one death in 2.02. These were all cases of traumatic tetanus. The percentage of death in the British army during the Crimean War was 91 per cent.; and Baron Larrey's estimate of mortality of the French army under Napoleon was at about the same rate.

In regard to the time of death Dr. Jones found that of 50 cases, in which the disease followed slight injury of the extremities, 43 proved fatal in a short time, and of the whole number of deaths reported 24.14 per cent. ran a rapid course after slight injuries, and terminated in death in a few days. One case died on the second day. Cases are reported which have terminated fatally in twenty-four hours after the appearance of symptoms. In one case, mentioned by Dazelle, they appeared on the third day, and the patient died the same night. One author lays stress upon the statement that the prognosis is governed by the interval that elapses between the receipt of the wound and the appearance of the symptoms, and that the longer this interval is the more favorable are the patient's chances. Many writers agree that elevated temperature plays an important part in the prognosis, and that any increase is to be looked upon with alarm. The duration of the attack is to be taken into account, and every day bridged over by the patient after the fourth or fifth increases his chances of recovery. Of course the gravity of the affection depends much upon the violence of the paroxysms.

**Treatment.**—It would be useless to discuss the merits of the many drugs that have been brought forward from time to time. Our most efficacious remedial agents are the depresso-motors, and among these may be mentioned chloroform, chloral hydrate, Indian hemp, Calabar bean, and conium.

Calabar bean, which has enjoyed a deserved popularity, has been made use of with great success by Eilert, Holhouse, Wood, Watson, and a host of others. Holhouse in 1864 reported two cases, one of which was cured after having taken 3–4½ grains of the extract every two hours. Ashdown was not so successful, and Spencer and Dickenson had the same discouraging experience. Even Watson was one of the first to use the remedy, and three out of his four cases of tetanus were cured by the administration of ten drops of the tincture every hour, and by a subsequent increase in the dose. The drug may be given in full doses, say from one-quarter to one-third of a grain of the extract every two hours.

The chloral treatment has certainly been more efficacious. Surgeon-Major Hunter<sup>2</sup> reported two cases: one a boy, and the other a man of 40. In the first case chloral was combined with *cannabis indica*. R. Tr. *cannabis ind.* ℥x; potass. bromid. gr. v, every third morning; and chloral hydrat. gr. xij, three times a day, together with inhalations of chloroform

<sup>1</sup> Medical and Surgical Memoirs, vol. i., New Orleans, 1876.

<sup>2</sup> Indian Med. Gaz., Feb. 1, 1875.



as required. The other patient took 20 grains of the chloral thrice daily. Opium and chloral in combination have perhaps been more effective than the chloral alone, and Delsal<sup>1</sup> saved three cases out of four by this treatment. H. C. Wood reports 9 cures by chloral out of 18 cases.

Chloroform has not proved to be the valuable remedy that many have supposed it to be, and it has only the power to "crowd down the bad symptoms which burst forth usually with additional fury when the narcosis subsides."

Aconite has been of service upon many occasions. It was first used by Page<sup>2</sup> in a case of traumatic tetanus. The toxic effects of the drug were produced, and during their continuance there was a remission of symptoms. The patient was first reduced to a condition bordering on syncope, and afterwards stimulated. De Morgan and others cured tetanus with this remedy, and its place in the therapeutics of the affection is by no means an inferior one. The pulse is markedly lowered, the muscular rigidity relaxed and a condition of akinesis and prostration takes the place of the irritable nervous state. Curare, nitrite of amyl. and belladonna, as well as a host of remedies of the same character, have been praised from time to time; but most of them are useless. Chloral hydrate, either in combination with aconite, or chloroform, and cold to the spine, which may be applied by the ether spray as recommended by Carpenter, I think is the best form of treatment, and should be resorted to as early as possible. If these remedies fail, Calabar bean, hyoscyamin, curare, or nitrite of amyl may be tried, and conium, which is a powerful depressor of spinal excitability, may be given a trial. Warm baths have been recommended.

"Dr. F. Franzolini<sup>3</sup> relates a case of tetanus arising from exposure by sleeping on the damp ground after great fatigue, successfully treated by prolonged warm baths and the continual use of chloral and morphia. The chloral was given frequently by the stomach, and the morphia by subcutaneous injection. The first bath was for six hours, at a temperature of 40° C. (104° F.), and subsequent ones lasted five, four, three, or two hours. This treatment was carried out from the 18th to the 30th of the month; but the daily use of chloral and morphia was continued some time longer. Of the first ninety hours of his disease, the patient passed forty-eight in the bath at 40° C. In twenty-nine days he consumed nearly four ounces of chloral hydrate, and about twenty-two grains of hydrochlorate of morphia were injected. Although kept so long in a state of almost constant narcotism, the mental powers of the patient were in no way affected."

H. de Renzi,<sup>4</sup> of Genoa, has spoken highly of the dark-room treatment. His patient was kept absolutely quiet. He ascribes the success to the

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<sup>1</sup> Quoted in Practitioner, August, 1877.

<sup>2</sup> Lancet, April 4, 1846.

<sup>3</sup> The Doctor, Oct. 1, 1875. Abs. in Phila. Med. Times, Oct. 30, 1875.

<sup>4</sup> Gaz. Méd. de Paris, No. 32, 1877.

belief that the absorption of oxygen and elimination of carbonic oxide are impeded by darkness.

The other indications seemed to be perfect quiet, and during and after the attack ample nourishment. Niemeyer<sup>1</sup> believes in clysters containing twenty or thirty drops of laudanum. He also recommends chamomile baths in the infantile variety.

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<sup>1</sup> Text-Book of Pract. Med., vol. ii. p. 352.

## CHAPTER XIII.

## BULBAR DISEASES.

## EPILEPSY.

**Synonyms.**—L'Epilepsie (Fr.); Fallsucht (Ger.); Mal caduco (Ital.).

**Definition.**—This most familiar of all nervous diseases is characterized by loss of consciousness of variable duration, attended or unattended by either slight muscular spasms or general convulsions.

The relation of these two elements, the psychical and physical, is not always the same, as in some forms of the disease there is a momentary loss of consciousness and perhaps no appreciable spasm, or the two may co-exist, there being protracted loss of consciousness and violent convulsions. There are sometimes very peculiar combinations of symptoms which will receive mention hereafter.

The modern investigation of epilepsy by Hughlings Jackson has materially modified our views of the disease. His consideration of the pathology of the disease is exceedingly complex, and he is inclined to treat the subject with greater breadth, and give it greater importance than it ever has received. A disruption of the most transient description of the harmonious relation of the psychical centres gives rise to a genuine paroxysm or discharge, so that many temporary *bizarre* actions which most of us indulge in even in comparative health, become invested with a new significance. Certain phases of what we indefinitely call "absent-mindedness," leading us to commit absurd acts which we laugh at after they are performed, may be in reality genuine epilepsies, and in others may attain the importance of disease symptoms.

The scope of this work does not permit me to consider the history of the disease; suffice it to say, that its antiquity dates back to the days of Hippocrates and Aretæus, and biblical references to its existence are common.

Cook<sup>1</sup> thus speaks of the early writings: "Epilepsy has been distinguished by a great variety of names such as morbus sacer, comitialis herculeus, caducus, etc. Aretæus says, it may have been called sacred on account of the magnitude of the evil, it being customary to call what is great by that name; or because it is to be cured rather by the Divine than by human power, or because persons laboring under it have been thought possessed by demons."<sup>2</sup> . . . Some of the ancients were of

<sup>1</sup> Treatise on Nervous Diseases, Am. ed., 1824, p. 326.

<sup>2</sup> Aret. de Caus. et Sign. Morb., lib. i. c. 4.



opinion that epilepsy was denominated the Herculean disease because Hercules was subject to it; but Galen says, it was so called on account of its form or magnitude."

"Epilepsy was denominated *morbus comitialis*, either because it frequently occurred in the crowded assemblies of the Romans called *comitia*, in which the passions of the people were often much excited, by which it might be occasioned, or because it was customary to dissolve the *comitia* if during the sitting any person should be affected by it.

"The application of the term *caducus*, a falling sickness, is too evident to need illustration."

In our description of the affection it is impossible to make any well-defined division; suffice it to say, that all writers recognize forms known as *Haut mal* or *Epilepsia gravior*, and *Petit mal* or *Epilepsia mitior*. Reynolds divides the latter into two varieties, viz.: 1st. A form *with* evident spasms, and another *without* evident spasms. Besides these, various irregular forms have been included, such as *masked epilepsy* and *hystero-epilepsy*.

#### THE GRAVE ATTACK.

**Symptoms.**—The most familiar variety is known as *Epilepsia gravior*, and it may be described as an attack expressed in four stages: 1st. A premonitory stage; 2d. Stage of convulsion; 3d. Stage of subsidence; and 4th. A stage of stupor, or "after-stage" (Reynolds). The first stage may often be absent, for in many cases there is a sudden *début*; but if such be not the case, the patient may have well recognized warnings which may be either psychical (mental or emotional), motorial, sensorial, or vascular, these latter being objective indications. Though these warnings are spoken of by many patients, it is almost impossible to rely upon their testimony, as the demoralization dependent upon the anticipation of the attack, or the short duration of such premonitory symptoms, is sufficient to prevent them from analyzing their feelings. It is, however, possible in many instances to collect information from a number of cases which shall be a basis for the general classification of premonitory symptoms.

Very often the attack will be immediately preceded by a vague dread, or an undefined fear of some impending trouble.

In one of my cases—a remarkably clever and intelligent young lady—there is a condition of exhilaration of spirit, and a mental activity which lasts for some hours. Although deeply under the influence of the bromide, she will come out of her apathetic state and chat with her friends upon all subjects in the most entertaining manner. Twitching of the eyelids or of the lower extremities, vertigo with rotatory movement, and tremor are examples of the disorders of the motility which occasionally precede the attack. Sometimes there is an elevated sensitiveness of the organs of special sense.

Hallucinations of hearing and visual hallucinations are not uncommon. One of my patients has often seen a fiery cross; and another refers to a

locomotive with a glaring headlight, which rushes upon him, while a third hears voices; and in two cases the patients say that they "smell smoke." Morbid sensations, which cannot be defined, are spoken of occasionally, and a vague sense of weight in the epigastrium, head, or some other part of the body is a frequent precursor of the attack. Occasionally the peculiar sensations begin at some remote part of the body, and seem to move rapidly towards the head; such phenomena are known as *auræ*. These *auræ* have been compared to the blowing of wind over the surface, the creeping of insects upon the skin, or the pricking of needles. They last but a few seconds, and are sometimes perceived, but not always. In the wards under my charge at the Epileptic Hospital, the patients sometimes have perceived the *auræ* in time to seek the nurse or attract the notice of the other patients. Careful investigation of twenty-nine cases resulted in the discovery that eighteen of them had a warning of some kind, four had none, and the rest gave us unsatisfactory answers. After a long process of condensation of statements, I find that seven had an *aura* starting from the epigastric region, two complained of constriction of the chest, seven had slight vertigo, and one had an *aura* starting from the extremities, and in one there was trembling of the right hand. Headache preceded the attack in four, and the "indescribable feeling" of the coming fit was alluded to by a number. In one remarkable case the first intimation of the attack was the violent jerking of the head to one side, and a species of vertigo. In another case the patient muttered incoherently for a full minute before the actual attack. A third case was equally curious. The patient, whose mental condition was good, would, without any apparent reason, attract the attention of persons about him by the repetition of the syllables "be-lub-be-lub, be-lub, lub, lub-a-lub, a-lub," pitching his voice in a high key, and gradually lowering the tone until the last part of his utterance was hushed and low, and then, after giving vent to a species of groan, he would become convulsed. Trousseau<sup>1</sup> calls attention to the "vascular prodromata." A local determination of blood may occur in the finger, for instance, causing it to swell, reddening the skin, and rendering it successively, within a very short time, red, and of a more or less deep violet color; or, again, the skin may become excessively pale after having been injected for some time. The swelling is real, not apparent; for rings previously easy suddenly become too tight for the finger. The only premonitory symptom may sometimes be an involuntary discharge of urine. It is difficult to distinguish this accident, however, and it is very liable to be considered a part of the attack, which it may be in reality. <sup>2</sup>Dr. Hughlings Jackson has made a contribution of the study of *auræ* with reference to localization. When the epileptic paroxysm is preceded by vertigo with apparent rotation of objects, the attack begins on the right side and indicates a cortical lesion of the opposite side. When the *auræ* consist in perception of odors,

<sup>1</sup> Clinical Medicine, Am. ed., vol. i. p. 75.

<sup>2</sup> "Brain," July, 1880.

or epigastric sensation, or when masticatory movements of the jaw occur, the convulsions begin on the left side.

*2d Stage (Stage of Convulsion).*—In many cases the first indication of the attack is a wild cry, which startles those about the patient. I have seen a soldier marching in procession throw up his gun and shriek so loud as to be heard half a block away, and fall to the pavement in a convulsion. This shriek is a psychical manifestation, and different from another form of cry which the patient may utter. This second variety is less noisy, and is produced by the forcible expulsion of air through the vocal cords which follows spasm of the thoracic muscles. It is more a species of groan. Simultaneously there is loss of consciousness, and the patient falls to the ground, and is agitated by tonic contraction of all the muscles of the body, but usually those of one side more than the other; so that his body is twisted and bent. The muscles of the neck are strongly contracted, while the face is generally distorted. The stronger contraction of some muscles than others draws the weaker side so that movements are produced which are not the result of clonic contraction, but rather an evidence of unequally expended forces.<sup>1</sup> Respiration stops, or there may be a long expiration, and then stoppage altogether for a few seconds. The pulse is now rapid and very small, a result, probably, of compression of the arteries by muscular masses, and the heart-beats are strong. At the end of a few seconds, and rarely after a minute, the convulsions become clonic, the patient throwing his arms about violently, or bumping the back of his head upon the floor. He is still unconscious, and may have evacuations from his bowels and bladder, or, as in some of the cases that I have seen, there may be an emission of semen. Reynolds calls attention to vomiting, a symptom which I have several times witnessed. The respiration now becomes labored and rapid, and there may be snoring. Froth collects about the mouth, which may be tinged with blood, as the patient sometimes bites his tongue or lips. The surface, which was in the first stage quite pale and cool, now becomes dusky, and of a dark livid color. The pupils may remain dilated as they were at the onset of the attack, or may be unequal. From my note-book I find that the following points were observed in the twenty-nine cases previously alluded to. In twenty-six the convulsions were quite general. In three the legs were more convulsed than any other part. In three the arms were especially agitated. In one patient the movements were confined to the left side. The cry was very piercing in five instances. In three there was only a moan or gurgling expiratory sound. Twenty-four of these patients bit their tongues. In twenty-three the pupils were wildly dilated. In two the dilation was not so marked. In four no appreciable difference was noticed. After the stage of tonic convulsion, which lasts a few minutes, the third stage is reached. In the large number of cases the attack may begin by local convulsive movements in the hand or in some of the muscles of the face. The thumb may be sharply turned in and the fist clenched—the convulsion then becomes general.

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<sup>1</sup> Reynolds.



*3d Stage (Stage of Subsidence).*—This is marked by a gradual return of consciousness. The patient may stupidly turn his head or look upwards, the eyes having a meaningless expression, and the balls oscillating slightly. He may strive to express himself, but only gives utterance to a series of unintelligible sounds. He may make some effort to rise, but finds it impossible to do so. His pulse is small and thready, or sometimes full and bounding, especially when the first two stages have been short. His eyes are injected, and his pupils either normal or contracted.

*4th Stage (Stage of Stupor).*—Exhausted by his attack, he falls into a sound sleep, which is so profound that he lies where he has fallen, and resents any attempt to remove him. The stupor may be so deep, however, as to make him unmindful of what is going on about him. His sleep lasts for several hours, and is characterized by snoring. If the patient recovers without the stupor, he is very irritable and cross. He complains of headache, or perhaps nausea, and vomits; and his pulse is irritable and irregular. Thompson<sup>1</sup> calls attention to the tracings obtained in epilepsy when the heart is healthy, and it is possible to obtain good results. He as well as Lorain found that the sphygmograph tracing exhibited a distinct dirotic notch.

In regard to the time of attack, two divisions have been made—*nocturnal* and *diurnal*. I have thought it best to make another, viz.: *matutinal*.

Perhaps *nocturnal* epilepsy is much more common than the other forms, for a great many patients never have attacks at any other time, while some may have them at all times, and a few only during the day. A large number are attacked just as they awaken; and I have met this form so frequently that I prefer to use the term *matutinal* for the attacks occurring between five and nine in the morning. The only sign of a nocturnal attack may be the evidence of involuntary passages of urine and feces, and sometimes both. Blood upon the bed linen as a consequence of tongue-biting is another indication, and the trouble which is required to rouse the patient is a third. Of forty-eight patients, fourteen had their attacks at irregular hours, seventeen had them at night only, five in the day, and twelve in the morning.

The patient may sometimes do himself bodily harm during the convulsion and Dr. Maury, of Memphis, has communicated to me the following two cases of dislocation of the bones during an epileptic paroxysm. This is a rare accident in epilepsy, although it is not uncommon in tetanus.

CASE I. A man from Holly Springs, Miss., was sent to Dr. M. in Dec. 1876. The patient was sixty years of age, a planter, and of good habits. About one year before, after eating his supper, he became ill and had convulsions. In the night he had fresh convulsions, and suffered considerably from pain in the right shoulder. The convulsions recurred at intervals of ten days. When he was brought to Dr. M. the

<sup>1</sup> West Riding Reports, vol. ii. p. 303.

shoulder was found to be shrunk, and the humerus dislocated and immovable.

CASE II. A lady from Alabama, during the menopause, was affected with epilepsy about two years and a half before the Doctor saw her. She was attacked at night with convulsions and pain in left hip. These attacks occurred at intervals of from two to four weeks before she was seen by a physician. Left lower extremity found to be shortened about two inches, femur evidently dislocated. Muscular contraction on outside of leg; toes everted, and thigh turned inwards. In this case no attempt was made to reduce the dislocation. Whenever she had convulsions there was pain in region of liver.

#### THE LIGHT ATTACK.

**Symptoms.**—The lighter forms of epilepsy are included under the head of *Epilepsia mitior*, and are attended by a very transitory loss of consciousness. There may be little or absolutely no spasm, and the attack may be so unpronounced as to escape the notice of those persons who may happen to be present. The patient may be eating at the time, and suddenly drop his knife and fork; or he may be engaged in some occupation, and suspend operations for a second. In one of my patients the only indication of the attack was the rolling upwards of the eyes. Another, a gentleman, when writing would stop for a moment and go on with his work entirely unconscious of any interruption. If walking, there may be a sudden loss of equilibrium, but he rarely falls. The face may be blanched or flushed momentarily, and the patient may suffer no bodily discomfort, but is sometimes restless, depressed, or low-spirited.

An aggravated state may exist, in which the muscular spasms are more marked.

The attacks, which have been described as “weak spells,” or “fainting fits,” by uninformed people, consist in more protracted loss of consciousness, accompanied perhaps by strong muscular contractions of the muscles of the face or arms, pallor, and dilatation of the pupils. I have a patient under observation who has a distinct epigastric aura; she then becomes rigid, holds her breath, grasps the arms of her chair; her head is drawn forwards, and so she remains for a minute or two.

The foregoing forms may coexist, there being distinct attacks of *grand mal*, with repeated *petit mal* seizures, which seem to have no special relation to the more serious convulsions. Twelve of the twenty-nine cases suffered from *grand mal* alone, and seventeen had both forms, and in these cases the *petit mal* predominated.

As to periodicity and frequency of the attacks there is much to be said. There is a peculiarity in the regularity of the seizures which is to be observed in very many cases. A tendency to weekly, semi-monthly, or monthly recurrence is noticed.

When the fits take place there may be only one at a time, or there may

be a number within twenty-four hours, or two or three days, and then an interval of the duration I have just described elapses before a fresh attack or series of attacks takes place.

In Reynolds's experience there are four times as many epileptics who have their attacks more frequently than once a month as there are who have them at long intervals; but I am disinclined to agree with him "that males are more subject to monthly attacks than females, and that attacks in the latter are not as a rule monthly seizures."

I discover every day numerous verifications of the menstrual influence. In forty patients I find that eighteen occur during or just after the days the woman has her catamenia; and in more than one case much interest arises from the fact that there was dysmenorrhœa, and that when this was relieved the attacks disappeared.

In many chronic cases, especially when there are complications, there is rarely any regularity in the appearance of the attacks. In the Epileptic Hospital, on Blackwell's Island, I find extreme variation in their number; and there are patients under treatment who have had but three or four attacks in one year, while there are others who generally have from five to thirty each week; but this great frequency is exceptional. The attacks of *petit mal* are much more numerous, but from their very transitory character it is difficult to make any estimate which is at all useful. The irregular forms of the disease are of greater interest as curiosities than anything else, but derive some importance from their medico-legal bearing.

#### IRREGULAR ATTACKS.

There may be a form known as *aborted epilepsy*, which consists in the expression of all the features of ordinary *grand mal*, without complete loss of consciousness. The attacks may occur in the course of ordinary epilepsy.

The most peculiar examples of irregular seizures are described by Falret, Hughlings Jackson, and others. While in certain states the patient will do the most eccentric things imaginable, the mind being apparently in a condition of vacuity, and the individual becomes more an automaton than a human being.

<sup>1</sup> Mesnet, of the St. Antoine Hospital, came across a very interesting case. The patient has been known as the "Automatic Man," and his history is as follows:—

"A young man during the late war had a portion of the left parietal bone, about eight centimetres in extent, carried away by a ball. Hemiplegia of the right side was the consequence, but this gradually disappeared. For some time past he has been the subject of attacks, lasting from twenty-four to forty-eight hours, attended by very extraordinary phenomena. During these he seems to act exactly like an automaton, walking continuously, incessantly moving his jaw, knitting his brow, and

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<sup>1</sup> *Gazette Hebdomadaire*, July 17, 1874.



appearing absolutely insensible to all that surrounds him. Not uttering a word, he walks straight forward, and when he meets with an obstacle, stops short, explores it with his hand, and tries to pass on one side of it. Surrounded by a circle of persons, he stops at each, and endeavors to pass by the intervals formed by their joined hands, then turns back, comes in contact with the next person, and resumes his round. All this time he never manifests the slightest consciousness, just as if he were in a state of somnambulism. He is absolutely insensible to pain, so that pins may be thrust through the cheek or into the fingers, or very powerful electric shocks may be administered without the slightest sensibility being manifested. What, however, is very remarkable, is that by bringing him in relation with certain objects we are enabled to determine in him the entire series of acts which are correlative with the sensation thus aroused. Thus, if a pen be placed in his hand, he seeks for ink and paper, and writes a letter in a very good hand, in which he speaks very sensibly about different matters which concern him. If a leaf of cigarette paper is placed in his hand, he feels in his pocket for the tobacco, rolls up the cigarette very adroitly, and, having found his match-box, lights it. If the match be extinguished just as it reaches the cigarette, he finds another, and that several times, until he is allowed to light his cigarette. If at the moment when the match is extinguished, another already lighted is presented to him in its place, it is impossible to induce him to light the cigarette by means of the substituted match. He allows his moustaches to become burned without offering any resistance, but he will not employ the light thus presented to him. If chopped charpie be placed in his pocket instead of his tobacco, he makes the cigarette with this, and lights and smokes it without seeming to pay any attention to what he is smoking.

Among the various experiments devised by Dr. Mesnet, there is one which is particularly curious. The young man is a singer at concerts by profession, and if gloves be placed in his hands he immediately puts them on, and searches for paper. When a roll of this, resembling music in form, is given to him, he places himself in the proper position and begins to sing. It would seem, in fact, that tactile sensation induced in him becomes the point of departure, and as if of escape, of a series of acts correlative to this initial sensation—acts which he accomplishes automatically, without letting them deviate from their habitual and regular succession. Lastly, it is to be noted that, while in this singular condition, the patient steals all that comes within his grasp. If he touches any person, he feels for his watch-pocket, and invariably detaches the watch and puts it in his own pocket, whence it may be immediately removed without his making the slightest opposition. The crisis once over, he has no recollection whatever of what he has been doing, and becomes again perfectly reasonable.”<sup>1</sup>

Equally curious cases are reported by Jackson of individuals who do purposeless things knowing nothing about them afterward. A patient of my own upon several occasions in a condition akin to “brown study,” walked from the ferry-boat into the wrong car and rode some miles before he discovered his mistake. Many of the curious cases of absent-mindedness reported by various authors were undoubtedly irregular forms of epilepsy.

<sup>1</sup> Med. Times and Gazette, July 25, 1874.

An irregular form of the disease is known as "masked epilepsy." The patient in this state may not fall to the ground, but while in a state of unconsciousness will evince a great deal of muscular activity. An epileptic in my ward is in the habit of tearing through the hall, colliding with such patients as may happen to be in her way, and finally recovering consciousness, when she has no recollection of her attack. I have noticed the same phenomena in other cases.

Another form is connected with the commission of purposeless acts such as I have cited. Cases of persons who have disappeared and travelled about the country for some days, and when found could not give the slightest history of their whereabouts are reported by various authorities. Such individuals in reality, lead a double life, and while the automatic state prevails they may commit deeds of violence which may subsequently cause a great deal of trouble; and in such cases only, the history of undoubted epilepsy should alone be sufficient to exonerate them. I believe it is strongly improbable that there is ever an attack of masked or aborted epilepsy without expression of some of the evidences of the true paroxysm.

The sequences of epilepsy are various, but it does not necessarily follow that any mental impairment should result. It is true that in some cases such a termination is possible. Idiocy and epilepsy sometimes go together, but it must be remembered that the former is a congenital state. Examples of general mental failure are by no means rare, and in some cases the disease slowly undermines the patient's intellectual condition. An apathetic state is the primary result. Any one who has seen one of these old cases (especially if the patient be the victim of *petit mal*), with dull fishy expression of eyes, dilatation of pupils, a leaden, sallow countenance, a full lip with imperfectly defined vermilion border, sluggish cutaneous circulation, loss of memory, and dulness of wits, will recognize the condition I have endeavored to describe. Dr. Gray,<sup>1</sup> of Brooklyn, has directed attention to what he believes to be a certain test of the epileptic state. He finds that the pupils of epileptics respond much more actively to the stimulus of light than in the normal individual. I cannot say that I have been struck with this condition. Dr. Gill, the Resident Physician of the Hospital for Epileptics or Paralytics, made an examination of the eyes of twenty-seven epileptics. Of this number, there was ready response in eighteen cases; of the remainder, seven responded slowly. In one other case the pupils were dilated, and responded only when a bright light was brought directly upon pupil. In the remaining case the pupils were contracted, and responded with great difficulty. Of fifteen cases, most of which were of recent date, the pupillary response was not remarkably rapid. The first eighteen cases were of long standing. In nearly all of these cases there was dilatation to a great extent under ordinary circumstances, and I attach much more importance to this appearance. When it is borne in mind that at best epilepsy is often a

symptomatic condition of various organic troubles which may affect the eyes in different ways it is difficult to attach pathognomonic importance to ocular tests.

An epileptic convulsion in infancy may give rise to cerebral hemorrhage from a vessel ruptured during the paroxysm, but the accident is almost unheard of in adult life.

Epileptic mania, which Reynolds considers to occur in about one-tenth of all the cases, is not confined to any particular time. It may occur before the attacks, or, as is more often the case, succeed them. In this condition epileptics may be occasionally very dangerous, and give way to outbursts of violence, for which, of course, they are entirely irresponsible.

A man who was a patient in the out-door department of the N. Y. State Hospital for Diseases of the Nervous System, and who had been treated by Dr. J. J. Mason, for epilepsy for a long time, was subsequently discharged, as it was supposed, cured. A month or two afterwards, having an attack which was undoubtedly epileptic mania, he pursued his wife through the streets, and, drawing a pistol, shot her through the heart. After the deed he expressed great remorse, and gave himself up to the authorities, but, notwithstanding the medical testimony, was sentenced to the State's prison for life.

**Causes.**—Of one hundred and eighty-three cases of epilepsy I have seen at various times, the ages at which the disease appeared were as follows:—

|                                   | Male.          | Female.   | Total.     |
|-----------------------------------|----------------|-----------|------------|
| Under 10 years . . . . .          | 16             | 10        | 26         |
| Between 10 and 20 years . . . . . | 23             | 48        | 71         |
| Between 20 and 30 " . . . . .     |                | 14        | 41         |
| Between 30 and 50 " . . . . .     | 29             | 11        | 40         |
| Over 50 " . . . . .               | 4 <sup>1</sup> | 1         | 5          |
|                                   | <u>99</u>      | <u>84</u> | <u>183</u> |

Hugon<sup>2</sup> has recently made a valuable addition to the literature of epilepsy in an excellent brochure upon the subject of etiology.

He gives a table prepared by Martinet to show the proportion of cases beginning between the 10th and 20th years.

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| Of 307 cases collected by Musset, there were . . . . . | 107 |
| " 68 " " Herpin, " . . . . .                           | 27  |
| " 83 " " Maisonneuve, there were . . . . .             | 46  |
| " 306 " " Alègre, " . . . . .                          | 105 |
| " 106 " " Leuret, " . . . . .                          | 42  |
| " 230 " " Moreau, " . . . . .                          | 76  |
| " 48 " " Dunaut, " . . . . .                           | 26  |
| " 70 " " Delasiauve, " . . . . .                       | 17  |
| " 75 " " Dussart, " . . . . .                          | 40  |

<sup>1</sup> In two of these cases there was an indication of syphilis.

<sup>2</sup> *Récherches sur les Causes de l'Epilepsie, etc.*, Paris, 1876.



It will therefore be seen that nearly half of all the cases begin before the twentieth year. Beau collected 273 cases, 43 of which began between the 6th and 12th years; 49 between the 12th and 16th years; and 17 between the 16th and 20th years.

The attacks of early life are exceedingly irregular, and may begin as poorly developed paroxysms, which are by many classified under that most convenient term *eclampsia*, which oftentimes means nothing. A number of these attacks of an undefined type usually precede the genuine explosion of the real disease.

In regard to sex, it may be said that Beaumès, Esquirol, and Moreau were of the opinion that the disease was more confined to women than men; but on the other hand Celsus, Joseph Frank, Leuret, and Sandras, as well as Reynolds and others, take the opposite ground. From the number of cases I have collected and tabulated, I am inclined to adopt the same view as the latter.

Of Hugon's<sup>1</sup> cases, 32 in number, 25 were men, and 7 women.

Professions seem to have very little to do with the production of the disease, if we except bar-tenders and liquor-dealers.

In regard to the predisposing influences of temperament, climate, and season, it has been shown by Foville, Marcé, Falret, and Delasiauve, that the nervous and sanguine temperaments predispose to the development of the disease. Maisonneuve found that of 65 cases, 25 were of a sanguine and 20 of a nervous temperament. Moreau considers that epilepsy is more frequent in winter than in summer, while others take the opposite view. Whether climate affects the development of epilepsy, I am unable to say; but, after very carefully conducted experiments in regard to the influence of temperature, I am prepared to state most decidedly that the attacks are much more frequent whenever there is a sudden change of weather.

A writer in the *Revista-Sperimentale*, of May or August, 1875, has given tables showing the influence of atmospheric changes, temperature, etc., upon the occurrence of attacks. Before that time I began a series of observations at the Epileptic Hospital. These, when compared with the accurately taken charts of temperature, barometric pressure, wind, etc., of the Health Department, conclusively prove the truth of the assertion I have just made. The number of attacks seemed to increase just at the change; and a very hot day, followed by a cool one, would show an increase of from ten to fifteen seizures among my patients during the cool day, and *vice versa*.

The influence of heredity is more strongly shown in epilepsy than in any other nervous disease, except it may perhaps be progressive muscular atrophy. In cases that I have seen the taint can be traced back for several generations either by epilepsy, neuralgia, insanity, or other nervous diseases. In one case the maternal grandfather died insane, the paternal grandfather died of apoplexy, the mother was living though subject to

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<sup>1</sup> Op. cit., page 7.

neuralgia, one brother had chorea, and the other had committed suicide in a fit of temporary insanity. Other examples are very much like this. Leuret<sup>1</sup> found among 126 epileptic cases that there was a history of hereditary epilepsy in seven cases. Beau's<sup>2</sup> experience was equally interesting. Of 273 epileptics, there was hereditary predisposition in 18 cases. Leech and Fox<sup>3</sup> fixed the proportion of epileptics in whom hereditary taint was found at 36.8 per cent., which, as far as I can judge, is no exaggeration. Reynolds<sup>4</sup> states that in the upper classes this hereditary predisposition exists to a much greater extent, but calls attention to the difficulty of obtaining information. I have often been disappointed in getting reliable information, for this "skeleton in the closet" is kept closely guarded. I have been repeatedly astonished to find how strong this element is in the higher walks of life. In one family I find a long succession of insane ancestors, idiot children, and dissolute progeny, which fully accounted for the transmission of the disease. It is a fact, however, that it does not follow that, because a parent has been epileptic, the offspring shall inherit the disease. Voisin found among 96 cases, 24 which followed hereditary alcoholism and phthisis. It is often due in the first instance to exciting causes, which, if removed, would probably be followed by disappearance of the disease.

As to exciting causes, I may enumerate bad habits, excessive venery, syphilis, and uterine disease, which last I believe *to be one of the most important of all causes in the epilepsy of women*. Fright, grief, anxiety, overwork, blows on the head, and other traumatisms, also enter extremely into the etiology of the disease; and the disorders of digestion and the exanthematous disease often play a part in its causation. Onanism is a very common cause; and of 24 male cases I have seen during the past year, this vice existed in 9. I may extract the following data from a paper in which I analyzed the chronic cases under treatment at the Hospital upon Blackwell's Island:—

One-third of these patients suffered from intercurrent diseases; two had advanced phthisis; several had nephritic disease; and a great many were anæmic. In regard to the complicating troubles, I find that twelve were subject to headache, two were hemiplegic (right), the epilepsy following the hemiplegia, two suffered from sclerosis (one locomotor ataxia, the other diffused cerebral sclerosis), and one was an idiot.

When we came to examine into the causes we found more difficulty than we anticipated. The intelligence and memory were much below par in all. Scarlatina and variola preceded the disease in two, syphilis in one. In nine the attacks were connected with menstrual irregularities and uterine disease (versions and flexions), two of these were masturbators (by

<sup>1</sup> Recherches, sur l'Epilepsie, Arch. Gén. de Méd., 1843.

<sup>2</sup> Archives Gén. de Méd., May, 1836.

<sup>3</sup> Manchester Medical and Surgical Reporter, quoted by Reynolds.

<sup>4</sup> Syst. of Med., vol. ii., p. 295.

confession), one of whom has been cured since the habit was broken. One case only was traumatic, four were congenital, and several gave absurd answers which were unsatisfactory. These are examples of chronic cases, and of course many are intractable.

**Morbid Anatomy and Pathology.**—The variety of morbid appearances that have been found from time to time give no satisfactory explanation of the pathology of this disease, and we will not enter extensively into their discussion. Spicula of bone growing into the brain-substance, thickened meninges, deformities, or depressions of the cranial bones, vascular anomalies, cysts, tuberculous deposits, softening, and a host of other changes have been observed. Some of these are important appearances which should not be dismissed too hurriedly. Undoubtedly the osseous changes are quite satisfactory causes. In three cases I found spiculæ or nodules of bone growing into or pressing upon the cerebrum. In one of these the exostosis had attained a length of one inch, and varied from one-eighth to one-quarter of an inch in diameter. In other cases I have seen decided depressions of the parietal bones, which impinged to a great extent upon the brain-substance beneath. As far as the deep lesions go, nothing very conclusive has been found. Van-der-Kölk has dwelt at length upon the increased vascularity of the medulla and the softened patches sometimes present, but these changes are just as likely to be the results of the disease as they are to be the lesion which produces the convulsion.

It seems likely, however, that the investigations of Cazauvieilh and Bouchet, Bourneville, Charcot, and Delasiauve in France, as well as those of Meynert in Germany, must throw some light upon the pathology of this puzzling disease. All of these observers found distinct induration of the cornu ammonis, or pes hippocampi. Cazauvieilh<sup>1</sup> reports eighteen autopsies made at La Salpêtrière. In nine of these one or both of the cornua ammonis were indurated, and at the same time there was induration of the white matter of the hemispheres. Bouchet,<sup>2</sup> in forty-three cases, found the same condition of affairs. He says, “La corne d’ammon est la partie cérébrale qui a le plus fréquemment présenté l’induration. Cette altération a souvent été si frappante, et quelquefois si constante, que bien évidente neuf fois de suite pour quelques médecins assistants, elle leur a donné la conviction qu’elle représentait exactement la cause pathologique de l’épilepsie.”

Bourneville observed this lesion five times out of thirty-four during the years 1866—1874. Meynert has repeatedly discovered induration of this part, and considers it a pathognomonic sign. In his examination the cornua ammonis were found atrophied, and appeared to be of a cartilaginous hardness, and had undergone a general alteration.

Of ten autopsies that I have made, six presented this lesion, and in one

<sup>1</sup> Archiv. Gén. de Méd., 3me Anné, 1825, i., ix., p. 510, et 4me Anné, 1827, i., v., p. 5.

<sup>2</sup> Sur l’Epilepsie (Annales Méd. Psychologiques, 1853, l. v., p. 209).



I found it to be uncomplicated. The other four cases presented nothing distinctive. In two the left hippocampus major was indurated, in three both were indurated, and in one the right was the seat of the same change. In one of these the extreme exterior part of the pes hippocampus was quite firm; the little crenations or irregularities were more marked than in the healthy brain, as there had evidently been some atrophy with contraction. In one the gray matter just adjacent to the hippocampus major contained several indurated patches. In two cases the veins which skirt the inner edge of the corpora striata at the line of the velum interpositum, and receive branches from these bodies, were quite distended with blood, as were the venæ galeni. The white matter in both anterior lobes was quite hard in three cases. In one case there were minute extravasations throughout the brain and in the medulla. In two cases there was effusion into the lateral ventricles. The cranial bones in one case were found to be considerably thickened. In all of the cases there were evidences of great meningeal hyperæmia. In three of these cases I found microscopical disorganization of a granular character of the nerve-elements in the medulla. The vascular walls were thickened, and at certain points ruptured, the places of rupture having no special pathological relation as far as the nuclear involvement was concerned.

In three cases which are not included in the ten referred to, I found osseous growths. Although this lesion of the cornua ammonis very rarely exists alone, it seems to be quite a constant morbid appearance, and it now remains for us to discover whether the condition is peculiar to epilepsy.

Pflüger<sup>1</sup> has made 300 autopsies at the Asylum of Ybbs, and in 25 cases of epilepsy, sclerosis of the cornua ammonis was found. The entire number of epileptics was 43. The cases in which their appearance was found to be most perfectly shown were those which dated from infancy. In three cases the disease did not begin until after twenty. Of twenty-three in whom the attacks were frequent and violent, seventeen presented this lesion. He supposes the alteration to be due to malnutrition following vascular trouble.

Epilepsy is, without doubt, an organic affection, the established disease beginning, perhaps, after a peripheral irritation has been transmitted repeatedly to the centres; but after the disease is fairly developed, the convulsions are not necessarily produced by the excitement of such distal irritation; for, as Nothnagel shows, in cases dependent upon a cicatrix the attacks are not, as a rule, excited only by irritation of the cicatrix. The clinical features of the disease prove the truth of this rule; for, in any well-established case, gastric, uterine, or any other reflected irritation may give rise to the seizures, or they may take place in an apparently spontaneous manner. We must, therefore, consider that epilepsy is a

<sup>1</sup> *Allegem. Zeitschrift für Psych. and Revue des. Sciences Med.*, 33, 1881, xxvi. p. 359.

disease of an organic character, expressing itself after either some distal or central stimulation in an irregular manner, or the result of both. That it is connected with central changes there is no reason to doubt; though these changes are by no means uniform.

The experiments of Brown-Séquard have thrown much light upon its pathology, though Nothnagel and others do not unreservedly accept his views.

The experiments of Brown-Séquard were chiefly made upon guinea-pigs. He produced epilepsy by division of the trunk of the sciatic, internal popliteal and posterior roots of the nerves innervating the lower extremities, and by injury of various parts of the brain, the corpora quadrigemina, and cerebral peduncles. He also divided the cord at different points partially or completely, and shows that injury of the lower part of the cord seemed to have more to do with the subsequent epilepsy than when the upper part was mutilated. After these experiments, the first appearance of epilepsy occurred in from four to six weeks. The attacks were either spontaneous, or followed irritation of certain parts of the skin which were included in the so-called "epileptic or epileptigenous zone." This included the cheek, anterior part and side of the neck, and a portion of the back. This region became anæsthetic, and the hair usually fell out. Any irritation of this tract, such, for instance, as pinching, gave rise to an attack. Ultimately the anæsthesia diminished, and the attacks subsided, so that it was impossible to excite them. The "epileptic zone" corresponded to the side upon which the nerve or cord injury had taken place.

Other forms of experimentation have produced convulsive attacks, or a condition resembling epilepsy. These were blows upon the back of the head (Westphall); irritation of the cortex-cerebri (Hitzig); ligation of the carotids and vertebral arteries (Cooper, Hall, Kussmaul, and Tenner); irritation of the peripheral sensory nerves (Nothnagel, Krauspe). The labors of these, as well as others, indubitably show that the epileptic attack is connected with cerebral anæmia, and the experimental production of this vascular state when irritation of peripheral sensory nerves has been made furnishes another link in the chain.

The question of localization next arises. Brown-Séquard, Schiff, Reynolds, and Kussmaul and Tenner have all demonstrated that the medulla oblongata is the probable pathological seat of the disease. It has been proved by them that a so-called "convulsive centre" is here located, which, when excited, by reflex stimuli, gives rise to extensive spasms of both kinds of the voluntary muscles; that whether the irritation comes *ex chorda* or *ex cerebro*, there is primary bulbar congestion, a cerebral anæmia, and a secondary cerebral congestion; that such congestion follows reflex spasm of the cervical muscles, and that a condition of venous engorgement ensues from pressure upon the large vessels of the neck. The pathology of the confirmed disease, as it has been generally considered heretofore, may be briefly stated as—

A. The existence of a condition of reflex excitability of the medulla from a long-standing reflected irritation.

B. An exciting impression transmitted from the periphery, or from a central part.

C. The irritation of the vaso-motor centre (described by Dittmar and others) through congestion at the floor of the fourth ventricle.

D. A secondary anæmia and hyperæmia of the hemispheres.

The production of symptoms probably due to—

1. *a.* Anæmia of the brain ; *b.* Consequential primary loss of consciousness, etc.

2. Irritation of “convulsive centre,” with tonic muscular contraction.

3. *a.* Irritation of nuclei of lower cranial nerves ; *b.* Consequential asphyxia. Contraction of muscles of neck, pressure upon vessels, etc., secondary stupor, clonic convulsions.

Van-der-Kölk<sup>1</sup> explains the tongue-biting as the result of irritation of the nuclei of the hypoglossal nerves.

The observations of Hughlings Jackson<sup>2</sup> and other modern observers throw much light upon the pathology, and give it a new and broader aspect. The former proves “that those parts are wont to suffer first and most which serve in the voluntary (special) operations, and those last and least which serve in the more automatic (general operations).”

Briefly to illustrate this, he quotes from an article in the *Lancet*, demonstrating that the three points at which the convulsions often begin are : “(1) in the hand ; (2) in the face, in the tongue, or both ; (3) in the foot.”

This confirms the idea that the onset begins in the parts devoted more particularly to the execution of voluntary movements. He has been enabled to prove that in this manner the parts first attacked are those which are more commonly affected in hemiplegia. He also calls attention to the phenomenon of aphasia, with epilepsy beginning in the right cheek.

“Epilepsies,” he says, “are the results of the second class of functional changes ; they are, speaking briefly, discharging lesions. But there are many varieties of discharges. Defined from the paroxysm, an epilepsy is a *sudden, excessive, and rapid* discharge of gray matter of some part of the brain ; it is a *local* discharge. To define it from the functional alteration, we say there is in a case of epilepsy, gray matter which is so abnormally nourished that it *occasionally* reaches very high tension and very unstable equilibrium, and, therefore, occasionally explodes. . . . It will be observed that the discharging lesion of epilepsy is supposed to be a *permanent* lesion ; there is gray matter which, since it is permanently under conditions of abnormal nutrition, is permanently abnormal in function. That this permanent abnormality is a varying state, has been said ; it has been remarked that the gray matter occasionally reaches

<sup>1</sup> Brain and Spinal Cord, Sydenham Trans.

<sup>2</sup> W. Riding Reports, vol. iii. p. 315, *et seq.*



high tension, and, therefore, *occasionally* discharges (or is discharged). There are waves of stability and instability. It follows from this that the first fit is supposed to be a discharge of a part which has *for some time* before been in a state of malnutrition; and a still further inference is that such 'causes' of epilepsies as fright are only determining causes of the *first* explosion. Many of the premonitory symptoms of a first attack are probably results of slight discharges; they are miniature *fits*."

That irritation of the auditory apparatus may give rise to a variety of epilepsy there can be no doubt, but such cases I believe to be rare. Brown-Séquard<sup>1</sup> states that Mr. Hinton, an English surgeon, has reported several where, after death, no lesion was discovered, except evidences of disease of the middle ear. My friend Dr. Roosa tells me that out of five or six thousand cases of aural disease he has seen, he does not remember but one of this kind:—This patient was under my observation.

John W. P——, aged 15 years and 6 months, a stout and apparently healthy boy, well nourished, and presenting no external evidences of disease; family history good. His mother stated that he had always been a rather dull boy, and that at school he was generally behind in his studies, and did not seem to learn easily, and when sent on errands, he was unreliable and forgetful. There is no history of injury or sudden fright, nor has there been any known predisposing or exciting cause; but at the age of eight years he had a severe attack of scarlatina, which left him with a remaining otitis, most severe on the right side, and resulting in a profuse discharge of pus, which still continues in a modified degree, but is not so excessive as it was a month ago. About six weeks ago he began to syringe his ears with a carbolic acid solution, which had the effect of removing a large mass of what was probably inspissated pus; and his hearing, which had before been quite defective, became greatly improved, and he no longer complained of various subjective noises, such as buzzing and roaring. When the quantity of discharge was diminished, his ears became painful, and pressure on the mastoid processes caused much suffering. Ever since the scarlatina he has had frontal and occipital headache, which is always constant. About a month ago he had his first epileptiform attack, and this occurred about noon one day when he was using his syringe. Without warning, he suddenly fell to the floor, became convulsed, and in a few minutes recovered, and did not fall asleep; but a semi-unconscious state, however, supervened.

The next attack came on four days after, at 3 P. M. While he was chatting with a friend, he suddenly stopped talking, and fell. This attack was much more violent than the first one. They now become more and more frequent, until about two weeks ago, when on one occasion he had fifteen during twenty-four hours. Since then he has not had so many, having had between one and five attacks every day but one, which was the only day he missed the attack since the commencement. During some of the attacks he is very violent, while in others not so much so. His appetite has been irregular for some time past. An examination made by Dr. Baldwin, House-physician of the Epileptic and

<sup>1</sup> Central Nervous System, p. 96, and Gaz. Méd. de Paris, 1842, p. 25.

Paralytic Hospital, and myself, revealed tenderness on pressure over mastoid processes, but mostly on the right side. He has had no definite aura, but peculiar sensations which he cannot describe, preceding his attacks. He complains of vertigo and nausea, and muscular weakness after the slightest exertion. He invariably returns to consciousness almost immediately after the attack, attempts to rise and walk, but is usually quite feeble.

*Examination of Ears.*—R.: Discharge scanty, thin, and sero-purulent; and, on examination, the membrum tympani is found absent. The tick of a watch is heard only when the watch is pressed against the ear; a roaring sound is always present.

L.: The same examination shows more or less congestion of the tympanum, with evident signs of otitis media; but there is not so much pain on this side, and the hearing is better, the ticking of the watch being heard at three inches.

Patient has complained lately of deep, severe pain in the frontal, but extending back to the occipital region. With this pain there is dizziness, especially when he stands, thus making it difficult for him to preserve his equilibrium, which is strikingly shown by his irregular movements. When sitting up in bed, he complains that objects move up and down, and not horizontally, as we should expect to find in ordinary auditory vertigo; and a very interesting and peculiar symptom are the movements he makes to preserve his relation with surrounding objects, his body moving up and down, and his head swaying strangely. He is very susceptible to noises and bright lights, either being capable of inducing a spasm at times. Vomiting from an empty stomach is occasional, with dilatation of pupils. The vision of right eye is at times entirely lost, but at others is unimpaired. *Muscae volitantes* are frequently complained of. Examination of urine affords negative results.

*Observations during an attack or convulsion, which occurs at no regular intervals, but is a constant result of irritation of the internal auditory apparatus:—*

Ear syringed at 9.55 A.M. Patient calm, and not at all nervous; skin of normal hue; pulse regular; temperature normal; pupils somewhat dilated. He passed a good night, and suffered but little pain, though his vertigo was still troublesome. He was placed upon a bed, and the point of an ordinary two-ounce syringe, filled with tepid water, was inserted in the external meatus of the right ear, and the contents gradually expelled. This caused some pain and dizziness, which increased as more water was injected; and when one ounce had been thrown in, the patient became suddenly unconscious, and the head was drawn from one side to the other by rapid clonic contractions of the muscles of the neck, and almost at the same time the convulsion became general, the muscles of the back being extensively involved.

About five seconds after this, there were clonic spasms of the muscles of the jaw, so that the patient snapped his teeth, and, at the same time, forcibly inspired, giving vent to a peculiar noise which might be easily compared, by a person of lively imagination, to the bark of a dog.

This paroxysm lasted two minutes, and during its continuance the pupils were widely dilated. The patient remained unconscious; but there was neither pallor nor suffusion of the face. Thirty seconds afterwards, a period of muscular relaxation succeeded, a fresh attack followed,

during which there was more marked opisthotonos, much more noise, but no frothing at the mouth. Pupils still dilated, though perhaps not so much so as at first, while the skin was slightly suffused; but there was no duskiess. Duration, one and a half minute. *Ten o'clock and thirty seconds*, after slight relaxation and subsidence of movements, the lateral jactitation of the head again began; and at *ten o'clock and one minute* a violent accession of clonic, and afterwards tonic spasms made their appearance. The eyeballs had throughout been uncovered, and at first were stationary and immovable, or almost so; but now they were agitated by nystagmatic movements, and the pupils were dilated. This paroxysm lasted but thirty seconds. At *ten o'clock and three minutes* there was another seizure, during which the left sterno-cleido-mastoideus was involved in a prolonged tonic contraction. The pupils now partially returned to their normal condition, which was one of slight dilatation; and at *ten o'clock and four minutes* the patient became semi-conscious, answered questions in monosyllables, and after a few minutes recovered entirely. The pulse suffered no variation, except, perhaps, after two minutes had elapsed from the beginning of the seizure, when it seemed to increase in volume, and perhaps slightly in rapidity. There was an entire absence of any external evidence of asphyxia, which is so marked in the more familiar form of epilepsy.

I have ascertained that the convulsions may be precipitated by simply blowing into the external auditory meatus.

**Diagnosis.**—Epileptic attacks may be mistaken for the convulsions of Bright's disease, infantile convulsions, hysteria, alcoholism, opium poisoning, syncope, and softening, and the disease is occasionally simulated by malingerers and others. I may briefly dispose of the above:

1. Uræmic convulsions are generally preceded by drowsiness or coma, delirium and stertor. The limbs may be œdematous, and the urine contain albumen.

2. Infantile convulsions from worms, dentition and other eccentric causes, are usually attended by a febrile condition. The convulsions are of short duration, and are characterized by complete loss of consciousness. The discovery and removal of the cause usually effect a disappearance of the attacks.

3. Hysteria (See article Hystero-Epilepsy.)

4. Alcoholism and opium poisoning are characterized by a more protracted stage of unconsciousness, and by a contraction of the pupils in the latter.

5. Fainting attacks may resemble the *petit-mal*, but there are no spasms, and the pulse is feeble.

6. Softening and other organic states give rise to convulsions, but the accompanying symptoms should enable the observer to make the diagnosis in every instance.

Simulated convulsions may deceive a careless person, but the normal condition of the pupil, and the eagerness of the individual to play his part perfectly which he does not do, lead to the detection of the imposition; and the excessive pallor of the first stage can never be simulated.



<sup>1</sup> Dr. Carlos Macdonald reports the case of a patient who feigned epilepsy and who was known as Clegg the "dummy chucker." Clegg was a criminal, and feigned epilepsy so successfully that he escaped hard work and was generally regarded by a number of prison physicians as an object of sympathy. He submitted to all manner of painful tests, and upon one occasion he actually fell twenty or thirty feet in one of his shammed attacks. Dr. Macdonald, however, was suspicious and watched him very carefully and finally compelled the man to confess. In his pretended paroxysm the hands were closed but the thumbs were not so closed, nor were they flexed at any time, and the sphincters were never relaxed. His facial expression at times betrayed him when he was closely watched. There was no lividity beneath his nails. These indications, together with the patient's manner, which was ostentatious, so far as showing his scars and alluding to his feelings was concerned, convinced Dr. Macdonald of the deception.

The syphilitic form of the disease resembles much the ordinary variety, but in some instances it is of the greatest importance to distinguish its specific nature, as of course the treatment is entirely different from that employed in the non-specific disease. Buzzard, who has given us an admirable little work on the syphilitic neuroses, lays great stress upon the necessity of recognizing the variety of pain as a differential symptom.

"If pain in the head be associated with convulsive attacks," he says, "it generally *precedes* the attack in syphilitic convulsions, and is often localized in one particular spot. . . . In simple epilepsy (if it be present) it almost always follows the fit, is diffused over the forehead, and is at no time a strongly marked symptom." The age of the patient, and the time from which the attacks date, are also of great importance in this connection. It is not probable that syphilitic epilepsy would begin early in life, or, at least, before puberty, but simple epilepsy dates from early childhood.

**Prognosis.**—The duration of the disease has much to do with the prognosis, and the mode of origin, form of expression, and complicating conditions must all be considered before an opinion is given. If the disease be of idiopathic origin, or if it be due to violence, *i. e.* injuries to the head, the prognosis is bad. If it be due to eccentric causes or syphilis, there is reason to be hopeful. Hereditary predisposition is an obstacle in our path which sometimes blocks the way to a cure. I have found that the *petit-mal* is also less amenable to treatment than the severe form, and that it is pretty sure to produce an impaired mental condition.

Reynolds thinks that the attacks which recur rapidly are more amenable than those which take place at long intervals, but this has not been my experience. If there be any considerable congenital lack of intelligence the case may be considered as incurable. The unfavorable conditions are the occurrence of a great many attacks in a short space of time, the biting of the tongue, and a condition which has been known as the

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<sup>1</sup> American Journal of Insanity, July, 1880.

"status epilepticus," in which the patient lapses into a comatose state, and there are a number of fits in close succession. Death in the actual fit is not common, and I know of but six fatal cases: five from the disease, and one from falling upon a sharp iron point which penetrated the orbit.

**Treatment.**—Before entering upon the discussion of particular modes of treatment, I desire again to refer to certain etiological facts which bear to a great extent upon the selection of remedies.

I may be pardoned for calling attention to practical points which may appear unimportant to some; but an experience gained from the management of a great many cases teaches me that they are to be carefully considered in selecting a plan of treatment. These simple indications, I am convinced, are too often overlooked even by painstaking and careful medical men. I allude to the necessity for discovering the exciting cause. I am every day made to feel that the idiopathic cases do not form so large a proportion as they were once thought to. With this belief I am satisfied that empiricism and routine management are bad methods. Any one who examines all his cases thoroughly will recognize the delicate shades in epilepsy, variations which are exhibited in other diseases presenting more pronounced and better defined symptoms; consequently there are evidences of pathological action, which are not always grouped alike, and therefore all cases are not to be treated in the same manner. I ascribe the moderate success I have had in the management of this disease to the recognition of these differences.

Not only may obstinate epilepsy result from masturbation, but it may be due to many diseases of women, and it is produced by eccentric irritations of various kinds, or by centric irritation, such as may be associated with toxæmia.

Sir Charles Locock<sup>1</sup> called attention to many cases he had treated where uterine irritation was the exciting cause; and I think others have had the same experience. In one of Locock's cases the patient was affected particularly at the menstrual periods.

Some of these peripheral causes are curious in the extreme. Through the kindness of Dr. Gibney, of New York, I was enabled to see a child who had accidentally injured her ear with her parasol, the brass tip of which remained for some time imbedded in the external auditory meatus. As a result, convulsions of an epileptic character were caused, and it was not until some time afterward that the foreign body was discovered and removed. In another case I treated, the epilepsy was unmistakably due to a bad habit the woman had of wearing a number of heavy garments about her hips, which produced some uterine change. When this condition of affairs was noticed, and the skirts removed, she immediately recovered. At the root of many epilepsies, as well as other neuroses, are reflex causes—the starting-point being the organs of digestion, or those contained in the pelvis. Of course the varieties of epilepsy of an idio-

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<sup>1</sup> Med. Times and Gazette, May 23, 1853.

pathic nature, or those caused by traumatism or organic disease, will defy the best efforts of the physicians.

In prescribing for our patient there are five indications to observe:—

1. Removal of exciting causes, if possible.
2. The diminution of exaggerated reflex susceptibility of the medulla.
3. Equalization of cranial circulation.
4. Abortion of paroxysms.
5. Improvement of general condition.

For the accomplishment of these, it is imperative that a judicious and discreet selection of drugs should be made; and among those which are the most effective I may mention:—

The Bromides: sodium, potassium, ammonium, calcium, lithium, iron

|                  |             |                     |
|------------------|-------------|---------------------|
| Chloral hydrate. | Strychnine. | Arsenic.            |
| Belladonna.      | Ergot.      | Amyl-nitrite.       |
| Digitalis.       | Mercury.    | Tri-nitro-glycerin. |
| Cod-liver oil.   |             |                     |

I have not classified these remedies, as it is unnecessary to do so; but will now say a word in regard to their usefulness.

No one drug can be declared a specific, as I am sorry to see has been done; and we must not be too eager to accept the sanguine results of certain over-enthusiastic authorities, and be governed thereby. I allude more especially to the almost universal use of the bromides to the exclusion of everything else, and also to their employment in quantities which often ruin the patients, or, at any rate, produce a condition of diminished vitality, which is inconsistent with any hope of success. Radcliffe's<sup>1</sup> idea in this respect is a good one: "There is reason to believe that the therapeutics of convulsion must be based upon the notion that vital power has to be reinforced, and not upon the contrary opinion." What the proper dose is has not been clearly settled by any one. There are neurologists who believe in toxic doses, and there are others who prescribe quantities which are almost small enough to be inert. In England it has been the custom to prefer the small doses. I have seen the prescription of a very distinguished general practitioner, who some years ago thought five grains of the bromide of potassium a sufficient dose; but this has now changed. Ringer<sup>2</sup> recommends from 30 to 60 grains in the day; Radcliffe,<sup>3</sup> 45 grains; Russell Reynolds,<sup>4</sup> 30 to 90 grains; Bartholow,<sup>5</sup> 30 to 240

Handfield Jones<sup>6</sup> remarks that there is a great difference in the tolerance of individuals in regard to the bromides—some persons not being able to stand five grains, while others will not be affected by doses of less than forty grains.

<sup>1</sup> Pain, Epilepsy, and Paralysis, p. 215.

<sup>2</sup> Handbook of Therapeutics, p. 92.

<sup>3</sup> Op. cit., p. 202.

<sup>4</sup> Op. cit., p. 323, vol. ii.

<sup>5</sup> Materia Medica and Therapeutics, p. 371.

<sup>6</sup> Functional Nervous Diseases, p. 325.



My own experience has taught me that the best effect can be gained by the repeated administration of sixty grains in the twenty-four hours. The larger doses produce rapid bromism, while the medium dose seems to be better appropriated, but will do just as much mischief in the way of bromism as the larger one, if given for a length of time. My records show me that the average time for development of symptoms of this kind is about three months, while anæsthesia of the fauces is produced in a few weeks, or even a much shorter time; and I agree with others that it is necessary to produce this condition before we can say that the medicine has produced its physiological effect. But when once reached, the further toxic action of the drug is deleterious instead of beneficial. Brown-Séquard considers the appearance of acne to be an indication that the medicine has begun to do its work, in which opinion he is joined by Dr. Putnam-Jacobi.<sup>1</sup> Voisin<sup>2</sup> considers the "point of saturation to be indicated by the anæsthesia of the pharynx and nares, so that in one case nausea is not produced by titillation with a spoon, and in the other sneezing and weeping do not follow the introduction of a straw into the nasal cavity." I should consider the latter a rather severe test. According to Danton,<sup>3</sup> the bromides act as vascular medicaments, diminishing excitomotor power. They act on the unstriped muscular fibre, producing local anæmia, and moderating excitation resulting from temporary or permanent congestion. "They are agents that pass very rapidly into the blood (Ringer),<sup>4</sup> and consequently their effects are very immediate, and they accumulate till the point of saturation is reached before they are eliminated in anything like considerable amounts." We are all aware that repeated and large doses of these drugs are followed by a most disagreeable and pernicious state of affairs. Voisin<sup>5</sup> has referred to two forms of bromism, which he has divided, into the slow and rapid. In the first the complexion becomes muddy, the eyes sunken, sight and hearing poor, and memory obscure. The patient cannot write, and cannot express himself, as he forgets words; there is tremulousness. In the other variety of the *slow* form there is dementia, or delirium with manical outbursts. Ataxia is also a feature of this variety. In the *rapid* form—that with which we are most familiar—somnolence, headache, uncertain walk, difficulty of speech, loss of expression, "fishiness" of the eyes, drooling of saliva, etc. etc., are the ordinary symptoms.

Various grades of toxæmia, or even a state which Voisin calls the "cachexie bromique," and which terminates in a typhoid condition, may result from a reckless use of this drug.

As regards the variety of bromide, I think the sodic is the most reliable and stable, the potassic salt varying very much in strength. The others

<sup>1</sup> Oral communication before Am. Neurological Association.

<sup>2</sup> Voisin, *Archiv. de Médecine*, Jan. 1873.

<sup>3</sup> Danton, *Thèse de Paris*, 1874.

<sup>4</sup> *Op. cit.* p. 91.

<sup>5</sup> Voisin, *Archiv. de Médecine*, Jan. 1873.

either have a tendency to deliquesce, or are expensive. It will be advisable to keep the solution in a tight-stoppered bottle, and have fresh quantities put up constantly, as it is very apt to undergo changes—in which the bromine is evolved. And now a word regarding the time of administration. It has been shown repeatedly that these salts are much better absorbed when the stomach is empty. I have found also that a heavy dose at night is apt to do more good than if the amount prescribed is equally divided up through the day. In a great many patients I have found the attacks to occur at the waking hour, and I suppose this is due to the sudden change in the cerebral circulation. A mild diffusive stimulant has overcome this, and in many cases warded off the attack. I direct my patients who have their convulsion at this time to keep a glass or a small quantity of spts. ammoniæ aromaticus near at hand, to be taken before rising. Cold douches to the head are valuable. If the attacks be irregular, it will be found necessary to divide the dose.

*Analysis of Eleven Cases of Epilepsy.*

S. B.—Sodic bromide.

P. B.—Potassic bromide.

| No. of cases. | Sex and age. | Duration of disease. | Average No. of attacks before treatment.   | Maximum dose.                 | Minimum dose.             | Diminution.           | Remarks.   |
|---------------|--------------|----------------------|--|-------------------------------|---------------------------|-----------------------|--|
| 1             | Male, 15     | Since birth          | 1-2 weekly                                 | S. B. gr. xx.<br>t. i. d.     | S. B. gr. xv.<br>t. i. d. | 2 in 8 weeks          | Weak intellect.  |
| 2             | Male, 22     | Two years            | 1-2 weekly                                 | S. B. gr. xv.                 | .....                     | 1 in 20 wk's          | Disease followed sunstroke; treatment lasted three months. |
| 3             | Male, 25     | One year             | 1 or more in week, sometimes many in a day | S. B. xxv.,<br>P. B. gr. xxx. | S. B. gr. ij.             | None in 8 weeks.      | Hard drinker, feeble intellect; potassium salt inert.      |
| 4             | Female, 2    | 18 months            | 1-2 weekly, sometimes 3 in a day           | Very small doses.             | .....                     | None in 8 weeks.      | Fits followed dentition; rickety constitution.             |
| 5             | Female, 18   | One year             | 1 in week                                  | S. B. gr. xxx                 | Gr. xx.                   | None in 4 weeks.      | Tuberculous disease.                                       |
| 6             | Male, 18     | Five years           | 4 in week                                  | S. B. -r. xv.                 | .....                     | None from 5 weeks.    | No affection of intellect.                                 |
| 7             | Female, 11   | Five years           | 2-3 in week                                | S. B. gr. xx.                 | S. B. gr. xv.             | 1 in 5 w'ks           | Followed a blow; subject to headache.                      |
| 8             | Female, 17   | Several months       | Sometimes 4-5 daily                        | S. B. gr. xv.                 | .....                     | None after treatment. | Has bitten tongue  |
| 9             | Male, 20     | 19 years             | 2-3 weekly                                 | S. B. gr. xl.                 | S. B. gr. xv.             | No fits for 2 weeks.  | No aura.   |
| 10            | Male, 13     | Two years            | 3 weekly                                   | S. B. gr. xxv                 | S. B. gr. xv.             | 1 in 3 w'ks.          | Well developed disease, facies epileptica well marked.     |
| 11            | Male, 25     | 11 years             | 1 in 2 weeks                               | S. B. gr. xx.                 | .....                     | 1 in 5 w'ks.          | No fits since beginning of treatment.                      |

By this table it will be seen that from fifteen to twenty grains of the sodic salt were required to immediately decrease the number of attacks.

The treatment of the disease in women should be directed as well to the pelvic organs. It will be found that the bromides will markedly affect the flow, and relieve the pain or uneasiness which is connected with the

menstrual period. Locally I have found that cold applied for a few minutes daily over the ovaries will modify the attacks should they be connected with irritation of any of the pelvic viscera. The progress of the disease should be soon modified by the doses I have recommended; and it will be seen by the table condensed from that prepared by Dr. Hollis,<sup>1</sup> that even smaller doses modified or cured the majority of the cases he cites. At the Epileptic and Paralytic Hospital, where most of the cases are the very worst that can be collected as regards chronicity, I find that sixty grains a day will cut short the attacks of a great many patients, and I have cured a number of private patients by this method. Dr. Hollis' cases were not selected, and are evidently hospital patients, like my own.

On succeeding pages will be found two tables. In one are tabulated the interesting features of twelve cases of epilepsy. They are old hospital patients, and had applied for admission after outside treatment had been exhausted. Even here the bromides, in the doses I have given, seem to do much for the sufferers. Head-injury and actual insanity make the prognosis as bad as it well can be, and treatment is simply palliative. Large doses have aggravated many of those cases.

The other observations are selected from my note-book, and are illustrative of the efficacy of the dose I have advocated. Bromism occurred in spite of all I could do in most of them, though it was a mild form and under control. The patients were all of the better class, and of course had all the advantages of comfortable homes, attentive friends, substantial food and good air, although many of them were inclined to over eating, as in fact all epileptics are. In this respect there is an advantage in favor of the poorer patients, who cannot obtain rich food.

And now regarding the large doses. If the idea is thoroughly to ruin the patient's health, enfeeble his mind, or perhaps drive him to an asylum, the toxic administration may be indulged in. It is very true that sometimes a rapid restoration may be brought about by "iron and quinine;" but there are many cases where the recovery is not quite so complete as one could wish for. Memory is enfeebled, and there is a cachexia which remains for an indefinite time. A darker side of the picture is not always displayed when brilliant results are detailed. This is the list of demented and those that have died. Dr. Janeway was present at the autopsies of two patients who died brominized, for certainly the examination disclosed no other cause of death. I myself have seen several demented cases, and I have no doubt others could tell the same story. I have used the bromides in combination with chloral hydrate, and have obtained the most excellent effects. Such good results as diminished condition of stupor and eruption, follow the administration of equal parts of chloral and the bromide of sodium. The bromides of ammonium and sodium with chloral as recommended by the N. Y. Therapeutical Society, may be employed.

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<sup>1</sup> British Medical Journal, July 1, 1876, p. 4.



*Analysis of Twelve Cases of Chronic Epilepsy.*

S. Br.—Sodic bromide.    P. Br.—Potassic bromide.    H. Br.—Hydrobromic acid.

| No. | Age. | Duration of disease. | Average No. of attacks before treatment. | Average No. of attacks during treatment. | Maximum dose of drug.       | Minimum dose of drug. | Former treatment. | Duration of present treatment. | REMARKS.                                     |
|-----|------|----------------------|--|--|-----------------------------|-----------------------|-------------------|--------------------------------|--|
| 1   | 28   | 16 years             | 1 monthly                                | 1 in 2 months                            | . Br. gr. xx. t. i. d.      | P. Br. xv. t. i. d.   | .....             | 6 months                       | Has taken H. Br. for 1 month—fits increased. |
| 2   | 25   | 12 years             | 6 monthly                                | 1 monthly                                | H. Br. 1 dr., t. i. d.      | .....                 | S. Br.            | 1 month                        | Melancholic                                  |
| 3   | 25   | 20 years             | 18 monthly                               | 3 weekly                                 | Ergot (for 1 month)         | .....                 | .....             | 6 months                       | When under S. Br. attacks were 3 in month.   |
| 4   | 27   | 14 years             | 4 monthly                                | 1 monthly                                | P. Br. gr. xx. t. i. d.     | P. Br. xv. t. i. d.   | S. Br.            | 1 month                        | Masturbates.                                 |
| 5   | 19   | 6 years              | 7 monthly                                | 1 monthly                                | S. Br. gr. xx. t. i. d.     | .....                 | P. Br.            | 4 months                       | Hysterical                                   |
| 6   | 20   | 19 years             | 2 monthly                                | 1 in 6 weeks                             | P. Br. gr. lx., daily       | S. Br. x. t. i. d.    | Chloral, etc.     | 6 months                       | Menstrual complications.                     |
| 7   | 22   | 10 years             | 21 monthly                               | 2 weekly                                 | P. Br. gr. lx., daily       | .....                 | .....             | 9 months                       |  |
| 8   | 22   | 6 years              | 7 monthly                                | 1 weekly                                 | P. Br. gr. xv. t. i. d.     | .....                 | .....             | 6 months                       |  |
| 9   | 18   | 4 years              | 8 monthly                                | 1 monthly                                | F. ext. erg. j dr. t. i. d. | .....                 | .....             | 4 months                       | Bad habits.                                  |
| 10  | 23   | 23 years             | 3 monthly                                | 1 in 2 months                            | P. Br. gr. xv., t. i. d.    | .....                 | .....             | 4 months                       |  |
| 11  | 29   | 27 years             | 5 monthly                                | 1 in 3 months                            | S. Br. xx. t. i. d.         | .....                 | .....             | 6 months                       | Bad habits.                                  |
| 12  | 25   | 12 years             | 6 monthly                                | 1 weekly                                 | P. Br. gr. xx. t. i. d.     | S. Br. xv. t. i. d.   | .....             | 3 months                       |  |

*Analysis of Four Cures of Epilepsy with Moderate Doses of the Sodie Bromide.*

G. M.—Grand mal.

P. M.—Petit mal.

| No. | Sex. | Duration. | No. attacks before treatment.         | No. attacks during treatment.   | After first year.                          | Average dose, etc.   | Duration of active treatment. | Continued subsequent treatment.                           | Variety of disease.        | Period under observation. | REMARKS.   |
|-----|------|-----------|---------------------------------------|---|--|--|-------------------------------|---|----------------------------|---------------------------|--|
| 115 | M.   | 10 years  | G. m. about 4 weekly; p. m. 10 daily. | For first 3 months, 5; for next 6 months none.  | 2 in 2 years                               | 20 grs. sodie bromide, t. i. d.; auxiliary treatment; ergot, arsenic, iron, etc. | About 9 months                | X-xx grs. sodie bromide at night; strict hygienic care    | G. m. at night             | 4 years                   | Bright boy; epileptic habit almost confirmed; intelligent parents, who aided in general management and discipline; apparently idiopathic origin. |
| 229 | F.   | 12 years  | 1 to 2 weekly; g. m.                  | After xx. grs. S. Br. 2 in a week, 3 in next 2 weeks, 1 in next 5 weeks, 2 in next month, 1 in next 4 months, 1 in next 9 months.     | 3 in 2 years                               | Cod-liver oil, sodie bromide, xxv. grs., t. i. d.; atropia hypodermically.       | About 19 months               | Gr. x., t. i. d.  | Mixed                      | About 4 years             | Over study; hysterical; no uterine trouble.  |
| 336 | M.   | 9 years   | 1 to 2 every 2 months; g. m.          | S. Br. gr. xx. at night; 3 in 7 months.   | 1 in 1½ yrs.                               | S. Br. gr. xx., at night.  | About 2 years                 | Gr. x-xx, t. i. d. sea voyage; cod-liver oil              | Nocturnal                  | About 3 years             | Business man; overwork; supposed blow on head; attacks excited by over-eating.   |
| 417 | F.   | 5 years   | About 2 monthly; g. m.                | S. Br. gr. xx., t. i. d.; ergot increased them to about 5 monthly; worse at menstrual periods; stopped ergot; fits about once a month | 1 in 2½ year; none to date. Sep. 20, 1876. | S. Br. gr. xx., t. i. d.; uterine treatment; syr. lactophosph. of lime arsenic.  | About 4 months                | Lacto-phos. of lime; S. Br. gr. v., t. i. d. for 6 months | Mixed; Generally matutinal | About 4 years             | Case undoubtedly dependent upon interversion of uterus; immediate cessation of disease when the position of this organ was rectified.            |

Belladonna and its alkaloids are of great value when the seizures occur in the daytime, or are of the variety known as *petit mal*. I have injected the sulphate of atropia in  $\frac{1}{4}$  gr. doses beneath the skin at the back of the neck with good effect, and have used it in the manner directed by Trousseau. In either way it should be administered until dryness of the throat is obtained, and should be given a patient trial. The property possessed by belladonna of blunting reflex susceptibility assures it a great advantage over other methods of treatment, when there are centres of irritation such as in gastric epilepsy.

In ergot we have a remedy which controls the cranial circulation much more readily than any drug with which I am acquainted. As the object is to diminish the congestion at the floor of the fourth ventricle, its combination with the bromides greatly increases the action of the latter. Ergotin may be given alone in the form of Bonjean's capsules.

To Tyrrell<sup>1</sup> belongs the credit of suggesting strychnine. He believes that this remedy controls excitation of the medulla oblongata. In one individual who averaged fifty-one attacks in a month, the number was reduced by the strychnine to eleven in two years. Handfield Jones does not favor the remedy, nor do others, although it has advocates in this country. In small doses it certainly does good; but I have found that in larger doses than  $\frac{1}{32}$  gr., *ter in die*, it rather aggravates the disease.

Arsenic is excellent, both for its anti-periodic and alterative action, and as an agent to relieve the acne. Clemens, of Frankfort, has lately advocated the bromide of arsenic, but in such small doses as to seem useless. He claims for it remarkable virtue when the disease depends upon idiocy, and appears in patients with deformity of the skull. He reports two cures.

Dr. Hughes Bennet<sup>2</sup> reports the results of the bromide treatment in one hundred cases of epilepsy. In over sixty per cent. of the cases decided benefit resulted, the attacks being prevented or aborted. In about thirty-five per cent. there was bromism, and the remainder suffered from general enfeeblement of mind or body, without much benefit so far as the relief of the disease was concerned.

Dr. Bennet's method of administration consisted of doses of thirty grains of the bromides of potassium and ammonium, in the proportion of two parts of the former to one of the latter, given with aromatic spirits of ammonia and water. The dose was always given when the stomach was empty. After two or three months the dose was diminished.

Where there is an irregularity of heart action, sluggish circulation, blueness or duskiness of the skin, I think digitalis is indicated; in fact, I generally use it in every chronic case. It is a drug well tolerated by epileptics, who can take it in surprisingly large doses.

An agent has been lately given to the profession which seemed all that

<sup>1</sup> Med. Times and Gazette, May and August, 1867.

<sup>2</sup> Br. Med. Journal, June 7, 1873, and Journal of Nervous and Mental Diseases, October, 1879, p. 770.



was needed at first, but which I am convinced is very much over-estimated, except as an abortant. I speak of the amyl nitrite. Drs. Weir Mitchell, Zeigler, and Alexander McBride, as well as several foreign writers, have praised it, and several cures have been reported. In epilepsy there seems to be a "habit (if I may use the expression) or tendency to periodicity. Amyl is well adapted to stop this, as is any other remedy of the same class. Crichton Browne alludes to the effects of this drug upon the *status epilepticus*. His patient had had a great succession of fits, and was at the point of death; the pupils were contracted to an intense degree, pulse 116, temperature 102°, with stertorous breathing. Voluntary movements and yawning were caused by inhalation of the amyl nitrite, and the patient subsequently raised his head, looked about him, and recovered. Dr. Browne relates ten other cases which were seen with Dr. Mierson.

Dr. C. Steckete<sup>1</sup> draws the following conclusions in regard to the action of this drug in epilepsy:—

"It exerts an important influence where the epilepsy is due to or connected with cerebral anæmia, for the reason that it 'anticipates the attack when there are prodromata; cuts off the attack when it appears; relieves symptoms due to interrupted innervation after the attack; and the attacks become less frequent.'" He also considers it injurious where the attacks are due to cerebral hyperæmia, for the reason that they last longer and become more frequent, and when either maniacal or convulsive, increase in intensity.

My own experience with amyl nitrite has clearly settled in my mind the fact that it has great virtues in cutting short or averting attacks, but that it has no permanent influence. Whether we can or cannot make the delicate distinctions of Dr. Stekete<sup>2</sup>, future clinical experiences I think must decide. Those who have used it say that it does good in a very limited number of cases; and it is a difficult task to decide which are to be benefited. I have tried it in every grade of epilepsy, and find in some of the worst cases, where the fits occur all through the day with very slight intervals, and even where there is time enough to be prepared, that it is often of no avail. It may be given inclosed in the little glass capsules invented by Dr. McBride, of New York, for hospital use, and for patients who are not intelligent, in alcoholic solution.

<sup>2</sup> Bourneville and d' Ollier have used the bromide of ethyl in epilepsy and hysteria, and have found that when it was inhaled during the tonic phase of the attack, it produced an abortion of the subsequent stages of the attack. My experience with the new anæsthetic given in solution in epilepsy was not encouraging—but it may be given by inhalation in place of amyl.

<sup>3</sup> Berland has used tartar emetic in doses sufficient to produce vomiting

<sup>1</sup> Abstract of thesis in Chicago Journal of Nervous and Mental Disease, April, 1874, p. 260.

<sup>2</sup> Gaz. Med. de Paris, No. 35, 1880.

<sup>3</sup> These de Paris, 1880.

with marked relief in cases of violent convulsive chorea, and it seems worthy of trial in congestive epilepsy.

I may be pardoned for bringing another remedy to the notice of the profession, and one that has never been used for this purpose. I allude to tri-nitro-glycerine. Its reputation is almost enough to intimidate the patient, but it is as powerful a medicinal agent as it is an explosive. The tenth part of a drop touched to the tongue is sufficient in a space of time which is almost inappreciable to produce a rapid cerebral hyperæmia. The face is flushed, the eyes become bright, and the temporal vessels throb, while at the same time there are marked sensations of fulness. It produces more lasting congestion than does amyl nitrite, is much safer, and I have found it to act better as an abortant than the latter. Any good pharmacist can prepare a solution containing one drop to ten of alcohol. This can be further diluted, so that ten drops of alcohol shall contain one-tenth of a drop of the nitro-glycerine solution. It may be kept safe in this way, for alcohol prevents its explosion. A dose of from a tenth to one drop of the decimal solution is sufficient in the majority of cases.

Last of all, it seems almost unnecessary for me to direct attention to that most familiar remedy, cod-liver oil, which is so valuable in all nervous diseases. Anstie treated a number of cases by cod-liver oil alone, and cured seven out of twenty patients put upon this plan of treatment. In all cases I am convinced that it is a valuable remedy which is not appreciated as it should be. I have witnessed its great virtues when the bromide cachexia was profound, and believe that it should always be used in delicate subjects. Picrotoxin, a remedy recently brought forward, I have tried, and consider valueless.

The subjects of diet and personal habits are very important ones—particularly as the stomach is so often the seat of irritations which are transmitted to the over-active centres. Beyond the question of over-eating, it has been found that a vegetable diet is better suited to this class of patients. Mierson, in one of the volumes of the *West Riding Reports*, publishes cases, and makes comparisons between those epileptics placed upon a meat and those upon a vegetable diet. The results pointed to the superiority of the latter. As the greater number of epileptics have inordinate appetites, the diet should be strictly regulated.

It is a good plan, I think, to combine the remedies I have alluded to; and I take the liberty of presenting a prescription I have used for several years:—

R. Strychniæ sulph. gr. j.  
 Fl. ext. ergotæ,  $\bar{z}$ iss.  
 Sol. potass. arsenit.  $\bar{z}$ ij.  
 Sodii bromidi,  $\bar{z}$ iss.  
 Tr. digitalis,  $\bar{z}$ ij.  
 Aquæ menth. pip. ad  $\bar{z}$ iv.—M.

Sig.—A teaspoonful before eating, in a half tumblerful of water

If the attacks be the form known as *petit mal*, I think either ergot or belladonna are our best agents. With either form of treatment it may be found often necessary to use auxiliary general treatment. The syrup of the combined phosphates, or the syrup of the lacto-phosphate of lime, is a good adjunct; and salt baths, cold head douches, regular food, early hours, and the breaking off of bad habits, will often cure the disease, even when it has lasted many years.

As a last resort, should continued medication prove useless, the actual cautery or a deep seton at the back of the neck will occasionally arrest these bad cases.

A variety of other remedies have been suggested (and the list of drugs alone would fill several pages such as this), but as most of them have been found inefficacious, I do not think it worth while to further weary the patience of my readers. Galvanism I find to have but little value.

## BULBAR PARALYSIS.

**Synonyms.**—Glosso-labio-laryngeal paralysis (Hammond); Glosso-laryngeal paralysis (Trousseau); Progressive bulbar paralysis (Erb).

In the year 1841 Duchenne<sup>1</sup> first called attention to a peculiar group of symptoms which were connected with progressive degeneration of the medulla oblongata; and some years later Trousseau<sup>2</sup> noticed it in his admirable lectures, and presented several cases reported by Davaine,<sup>3</sup> long before Duchenne's observations were published, but which were before considered to be examples of double facial palsy. Hughlings Jackson,<sup>4</sup> Duménil,<sup>5</sup> Charcot,<sup>6</sup> and Joffroy, and lately Dowse,<sup>7</sup> have contributed to the literature of the subject.

**Definition.**—The condition under discussion may be described as a disease characterized by gradual loss of functions of parts supplied by the nerves taking their origin from the medulla, though the fifth nerve is rarely affected.

It may be the result of morbid changes which are limited to the floor of the fourth ventricle; or, this region may be the chance site of sclerosis, which affects other parts as well. Such may be the lesion, whether "pseudo-bulbar paralysis" (the result of arterial occlusion) sclerosis, or glosso-labio-laryngeal paralysis exists; the special symptoms are alike, and they appear one after another as the different nerves are involved.

<sup>1</sup> Op. cit., 2me edit.

<sup>2</sup> Lectures on Clinical Medicine, trans., vol. i. p. 908.

<sup>3</sup> Quoted by Trousseau, vol. i. p. 909.

<sup>4</sup> Philosophical Transactions, part i., 1868.

<sup>5</sup> Gaz. Hebdomadaire, June, 1859, p. 390.

<sup>6</sup> Archives. de Physiol., etc., tom. iii., 1870, p. 247.

<sup>7</sup> Brit. Med. Journ. Nov. 4 and 11, 1876.



**Symptoms.**—The earliest expression of the disease is a certain loss of power of the lips; the lower lip especially. If the individual attempts to whistle, his efforts may be unsuccessful, and the lower lip hangs so that the mucous surface is largely exposed. The tongue next follows, and its protrusion by the patient is a matter of difficulty. The individual is unable to bring the tip in contact with the roof of the mouth, and incompetent to use it in the formation of certain consonants (the linguals). When he tries to speak or read aloud he finds great difficulty in pronouncing words containing the letters l, n, c, d, g, h, j, t, w; and in one of Trousseau's cases the patient could not utter any letter but *a*.

He may remain in this condition for some time—say for a year or two, when the tongue and lips become more extensively affected; and not only are acts of a voluntary character impossible, but the automatic movements of the tongue are almost totally embarrassed. The use of this organ in the management of food during mastication and deglutition is much impaired, and particles of food become lodged between the teeth and the gums and cheek.

The patient's mouth is generally open, so that his teeth are exposed and from either side trickles a glairy stream of saliva. Next he cannot articulate the labials, and consequently his speech becomes worse than ever.

He wears an inane expression, and is apt to attract the attention of people in the street by his open mouth and silly appearance. The condition of the tongue has been noted by Dowse; its papillæ become atrophied, and the surface very smooth. I have noticed that there is no loss of the sense of taste at any time.

The palate next becomes the seat of the paralysis, and the pharyngeal muscles are so weak that deglutition is at first difficult, and finally impossible. Fluids are especially troublesome to swallow, and are apt to be regurgitated through the nares, and the voice becomes nasal and metallic as the upper part of the vocal apparatus becomes involved.

The facial expression, always a marked feature of the disease, is now very pitiable. The tongue lies in the bottom of the mouth utterly devoid of power, so that the patient cannot protrude it, and it becomes useless for all purposes. If the posterior wall of the pharynx be irritated, there is none of the reflex response which is so marked in the normal state, but only pain is produced. Such was the condition of affairs noticed in one of Dr. Dowse's patients.

The epiglottis does not cover the larynx; and there is a tendency to choking from the accidental introduction of food, so that eating becomes a dangerous undertaking. The voice grows very weak, and the sufferer can no longer even make the almost unintelligible sounds which characterized the early stages of his disease.

His breathing now becomes very irregular, the inspirations are quite slow and shallow, and he sinks from sheer exhaustion due to insufficient nourishment and becomes a mere wreck, dragging himself about, and looking forward to death as something which alone is to bring relief. As the

neumogastric becomes more and more involved, the respiration undergoes changes which result in asphyxia.

For some time before the end, his sufferings grow intense. Mucus collects in the bronchi, which he is unable to remove by coughing, and he sits in his chair with a feeling of greater security than when lying down, for in the supine position the saliva finds its way into the larynx, and produces suffocation. Loss of consciousness or mental impairment is never a symptom of the disease unless it be of the complicated form.

The following interesting case was reported recently by Dr. A. H. Smith,<sup>1</sup> of this city:—

The subject was a clergyman, aged sixty-one years. About fifteen years ago, after prolonged and severe exercise of the voice in preaching, he became hoarse, and ultimately his voice failed so that he could speak only in a whisper.

After the lapse of a year he gradually regained the use of the larynx, but as he did so he became sensible of an imperfection in his enunciation of certain syllables, especially those containing the letters p, t, d, s, etc. This difficulty has increased until now the power of uttering the labial and lingual sounds is almost entirely lost.

Later a difficulty in swallowing was gradually developed, which has reached such a degree that only *warm fluids* can be taken, and these with great care and hesitation, as they are apt to cause strangling, and to return through the nose. Mucus accumulates in the fauces, which he has great difficulty in getting rid of, and which causes a sense of strangulation.

He finds that the movements of the tongue are very much restricted, and he has not the full control of his lips.

His sight, taste, and smell are as perfect as is usual in persons of his age. The sense of touch, even in the paralyzed parts, is not impaired.

He feels much less distress when the weather is warm, and dreads the approach of each winter.

Such is the account which the patient—a very intelligent man—gave of himself. As to the objective appearances, the patient moved slowly and feebly, but this was evidently the result of mere debility. The next notable thing at a cursory glance was the expression of his mouth. The orbicularis muscle was entirely paralyzed, permitting the lower lip to fall away from the upper, and to become partly everted. There was also relaxation and eversion of the upper lip from the same cause. The levatores menti and the depressores ang. oris were not involved in the paralysis, and by their aid the patient was able to bring the lips into contact; but when so approximated they projected forward, leaving a space between them and the teeth, and giving a very peculiar expression to the face.

When the mouth was opened the movements of the tongue were observed to be very slow and very much restricted. The tip could not be turned upward to touch the roof of the mouth, nor backward beyond the bicuspid teeth. The tongue was not notably changed in shape or size.

All the muscles of the soft palate, including the palato-pharyngi and palato-glossi, were paralyzed, so that when the head was thrown

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<sup>1</sup> Med. Record, Nov. 24, 1877.

backward the relaxed velum fell of its own weight against the posterior wall of the pharynx. The finger carried into the fauces produced scarcely any local reflex action, showing that the constrictors were complicated; but sensation was perfect, and the reflex action of the stomach seemed unimpaired, efforts at vomiting being readily excited.

There was a very profuse secretion of mucus from the larynx and pharynx, which was gotten rid of with the utmost difficulty. There being perfect inability to contract the cavity of the pharynx, the air which was forced from the larynx in the act of hawking escaped into a great loose bag, instead of into a narrow, firm passage, and thus it failed to drive the mucus before it. The paralysis of the soft palate added to the difficulty, for when by great labor a portion of mucus was coughed up into the back part of the mouth, the non-closure of the isthmus faucium permitted it to fall back again upon the larynx.

Examination with the mirror showed that the laryngeal muscles retained their activity, and the cords, with the exception of slight hyperæmia, were normal. The respiratory muscles were as yet unimpaired.

In this case it is not probable that the loss of voice, which occurred in the early stage of the disease, was owing to a central lesion, since, after a year had passed, the larynx gradually regained its power. Moreover, laryngeal paralysis of bulbar origin does not usually occur in this association until after the paralysis of the lips, tongue, and soft palate has become well-marked. It is more than probable that the aphonia was the result of a catarrhal affection, and that if life continues long enough, there will be a return, but this time from advancing change in the medulla.

The greater ease in swallowing *warm* fluids is characteristic of dysphagia from almost any cause. Thus it is observed in both organic and spasmodic stricture of the œsophagus, and also when dysphagia results from the pressure of a tumor.

Dowse<sup>1</sup> considers the disease to be either *progressive*, *stationary*, or *retrogressive*, and if it were not for the single case of the last variety, which he publishes, I should not be prepared to accept the two latter divisions. This he calls *reflex bulbar paralysis*. His patient, a woman aged 59, suffered from Bright's disease and inflammation of the maxillary and parotid glands. After her recovery from the last-mentioned condition, there was paralysis of the hypoglossal, facial, and spinal accessory nerves, as well as the third division of the fifth. The vocal cords acted feebly, and she could scarce speak in a whisper, being able to pronounce only the linguals *r* and *s*, and could not protrude her tongue; food lodged in the cheeks; saliva dribbled from the mouth; she was unable to blow out a candle, while deglutition was interfered with to some extent. Strange to say, there has been improvement. It would be well, however, if Dr. Dowse had allowed a longer time to elapse before coming to a conclusion in regard to the retrogressive character of the disease in this instance, for the parotitis may have been simply a coincidence. I am inclined to think that the history of any genuine case thus far reported has shown a tendency to progressive decline, which, though delayed in some instances, has nevertheless steadily advanced to a fatal termination.

<sup>1</sup> Brit. Med. Journ., Nov. 11, 1876, p. 615.



**Causes.**—The disease is one of middle age, and attacks men more often than women. It is usually the result of syphilis, and sometimes follows exposure and mental worry. Dowse considers the causes of the peripheral symptoms to be the following:—

*Direct.*

1. Progressive interstitial neuritis.
2. Thrombosis.
3. Hemorrhage.
4. Morbid growths.
5. Vascular spasm.

} Rare.

*Indirect.*

1. Reflex action from peripheral irritation.
2. Inhibition from shock to central cerebral ganglia.

**Morbid Anatomy and Pathology.**—Trousseau's autopsies revealed induration of the medulla, atrophy of the roots of the hypoglossal and spinal accessory nerves, thickening, and gray discoloration of the dura mater on a level with the medulla, which extended as far down as the roots of the fourth cervical pair. "This thickening was due to a considerable increase in the amount of fibers of connective and fibro-elastic tissue, and seemed to result from a chronic congestive process, as shown by the great number of capillaries and of deposits of hæmatin external to them. The motor nerve-roots of many cervical nerves were found thinner than they should be from disappearance of nerve-tubes. The fifth and glosso-pharyngeal nerve-roots were healthy, and the muscular tissue of the paralyzed parts was found to be normal."

Duménil published a case which was probably progressive atrophy; but some of the symptoms were those of the disease under consideration. In this case there was extensive atrophy of the roots of the hypoglossal, pneumogastric, and facial nerves, as well as a great many other changes.

Fox<sup>1</sup> considers an absolute or partial disappearance of the nerve-tubes, with preservation of the neurilemma at the nerve-roots, to be a constant lesion; and Wilks<sup>2</sup> found that the roots of the hypoglossal and spinal accessory nerves had undergone atrophy, and become reduced to "little thin gelatinous threads."

Sclerosis may occasionally involve the medulla, and produce symptoms characteristic of loss of function in the nerves to which I have alluded.

Charcot<sup>3</sup> gives, among other cases, one that involved the medulla extensively. A patient of his presented, besides the ordinary symptoms of disseminated sclerosis, three months afterward, evidences of invasion of the pneumogastric and hypoglossal nerve-roots. There were dyspnœa and dysphagia. The patient was obliged to eat more slowly; and oftentimes the food was regurgitated through the nostrils. Death followed in about six weeks afterwards, and was preceded by asphyxia.

<sup>1</sup> Op. cit., p. 234.

<sup>2</sup> Guy's Hosp. Rep., vol. xv.

<sup>3</sup> *Léçons sur les maladies du système nerveux*, Paris, 1872-73. Première partie, p. 234.

The autopsy revealed the following state of the nervous centres: A section made one centimetre below the protuberance, at the point of origin of the trigeminus, disclosed a point of sclerosis. Other transverse sections were made at the smaller part of the olivary bodies, and a sclerosed patch was discovered. Another patch was seen at the root of the pneumogastric. Examination by the microscope revealed a number of broken nerve-tubes and broken-down cells at the nuclei of the hypoglossal, and traces of irritation in the white substance of Schwann in the pneumogastric fibers. The pharynx and larynx were healthy.

The observations of Lockhart Clarke have shown the intimate relationship of the nuclei of the important cranial nerves which become affected in bulbar paralysis. There is a set of nerve-cells common to these nerves, and disease of the nuclei of one nerve is very likely to extend to others of the group, so that ultimately there is a general invasion, which is bilateral and never one-sided.

The destructive process is probably myelitis, as Leyden has suggested, and disappearance of the motor-cells is the direct cause of the paralysis.

It is a curious fact that the sixth nerve invariably escapes when we remember that it arises from a common nucleus with the seventh, as demonstrated by Lockhart Clarke and Stilling. In regard to the partial paralysis of the facial as an early symptom, and the subsequent increase in the area paralyzed, we must remember Romberg's statement that in organic brain-disease the entire distribution is not affected, but that the fibers involved are those that supply the muscles of the upper lip and ala of the nose; and this is an important point in the diagnosis from peripheral paralysis; and Dowse calls to mind the fact that bilateral paralysis of the muscles supplied by the facial is connected with lesion at the root of the nerve.

The aphonia may result, according to Duménil, either from paralysis of the thoracic muscles, or those of the larynx. The ptialism I am inclined to ascribe, in the later stages, to paralysis of the chorda tympani, but agree with others who have observed it, that the accumulation of saliva in the first stage is due more to the patient's inability to swallow it than to anything else. Respiratory troubles may be due to paralysis of the pneumogastric and its motor, the spinal accessory.

Dowse has divided the disease into three stages as regards the difficulty of swallowing, the first of which is connected with paralysis of the hypo-glossal; the second with paralysis of the motor branches of the glosso-pharyngeal; and the third with paralysis of the spinal accessory.

Voisin, in speaking of the alterations in speech, defines them into stuttering, drawling, hesitation, jabbering, stammering, and quavering. The first three are due to lesions of the nerve-tracts which pass from the anterior cortex to the medulla oblongata, and which traverse the corpora striata, crura cerebri, and pons, and are connected with disturbances of will. The other three have no such origin, but depend upon inco-ordination of the muscles supplied by the hypoglossal, facial, and glosso-pharyngeal nerves.

**Diagnosis.**—Facial palsy, general paresis of the insane, progressive muscular atrophy and diphtheritic paralysis may suggest themselves, and some are rather difficult to exclude, among them tumor, which however is often attended by convulsive attacks:—

1. Facial palsy may be suggested, but as this disease is of sudden origin, and affects other muscles than those about the mouth, there need be no reason to confound it with bulbar paralysis.

2. The early symptoms of general paresis of the insane somewhat resemble the initial symptoms of the disease of which we are speaking. There is tremor of the tongue, however, in addition to the embarrassment of speech; contracted pupils and subsequent psychical symptoms make the diagnosis clear.

3. Progressive muscular atrophy, rarely attacks the tongue primarily, and only one case has been reported (by Charcot) where there were any bulbar symptoms. The subsequent atrophy of other muscles will dispel any doubts the observer may have. The affection of the medulla is ordinarily a final result of the extension of the central disease in progressive muscular atrophy.

4. Diphtheritic paralysis is symptomatized by initial paresis of the muscles of the pharynx, and the tongue is seldom involved. A previous history of diphtheria will confirm the cause of the paralysis, should there be a suspicion.

**Prognosis.**—As I have said, Dowse believes that there are forms of the disease which may be cured, viz., the *stationary* and the *retrogressive*. I cannot believe that when once affected by inflammatory disease, such extensive alteration, and such decided symptoms as he mentions, can ever be removed.

The histories of the cases reported by the several observers already mentioned certainly offer a gloomy prospect and little encouragement for the victim. The only case reported as actually cured was that of Cheadle,<sup>1</sup> and from the pain, visual trouble, and unilateral paralysis, it is improbable that the case was one of genuine bulbar paralysis.

Raynard<sup>2</sup> reports a case of bulbar paralysis with violent heart dilatation, syncope and speedy death. The heart was found after death to be greatly increased in size, and though its valves were unaffected, there was very decided dilatation of all the cavities.

**Treatment.**—Nothing has been done which has resulted in any decided improvement. I am sorry to say that electricity did no good in the one case I have treated, but Duchenne<sup>3</sup> in several cases found that systematic faradisation greatly facilitated articulation and otherwise helped his cases. Erb and Benedikt were particularly successful. Dowse recommends cod-liver oil, iron, and phosphorus, but Erb does not believe in the latter.

<sup>1</sup> Labio-glosso laryngeal Paralysis, St. George's Hosp. Reports, vol. v., 1871, p. 123.

<sup>2</sup> Quoted by Pitres in his Thesis, 1878.

<sup>3</sup> De l'électrisation, etc., 2d Ed. p. 649.



## CHAPTER XIV.

## CEREBRO-SPINAL DISEASES.

## CEREBRO-SPINAL MENINGITIS.

**Synonyms.**—Spotted fever; *Méningite foudroyante*; Head pleurisy; Myelitis petechialis; Cerebral or Cerebro-spinal typhus; *Méningite cérébro-spinale*; *Fièvre cérébro-spinale*, etc.

**Definition.**—A disease characterized by inflammation of the meninges of the brain and cord, symptomatized by pain, tetanic spasms, and herpetic eruptions, and occurring in an epidemic form.

This most terrible disease has of late years received a great deal of attention at the hands of German and French writers. Niemeyer<sup>1</sup> was one of the first of the former to direct attention to the disease; while in France, Broussais and others wrote extensively. There is no doubt as to the antiquity of the disease, for among the writings of Hippocrates a nearly perfect description of the malady is to be found. In our own country the epidemic character of the affection was noted by several of the older authors, among them North<sup>2</sup> (1811), Gallup<sup>3</sup> (1815), and Minor<sup>4</sup> (1823), and their contemporaries. Outbreaks occurred at Medfield, Mass., Litchfield Co., Conn., and at various points in the Eastern and Middle States during the early part of the present century. Clymer,<sup>5</sup> Jones,<sup>6</sup> and others have since written exhaustively on the subject.

Cerebro-spinal meningitis is certainly an irregular disease; it is not contagious, and is influenced seemingly in no way by climate.

**Symptoms.**—The appearance of symptoms is usually quite sudden, and their course is remarkably rapid and ordinarily tends to a fatal termination. In exceptional cases pain in the back, headache, vomiting, or malaise may constitute a premonitory stage, which lasts a few hours; but usually there is no such delay. A severe rigor, an attack of vomiting which is followed by headache of an intense description, and an elevation in pulse and temperature mark the commencement of the trouble. The child may present these symptoms, and in addition another which is invariably pathognomonic.

<sup>1</sup> Treatise referred to in Niemeyer's Text-Book of Prac. Med., vol. ii., p. 218.

<sup>2</sup> Treatise on a Malignant Epidemic, etc., 1811.

<sup>3</sup> Sketches of Epidemical Diseases, etc., 1815.

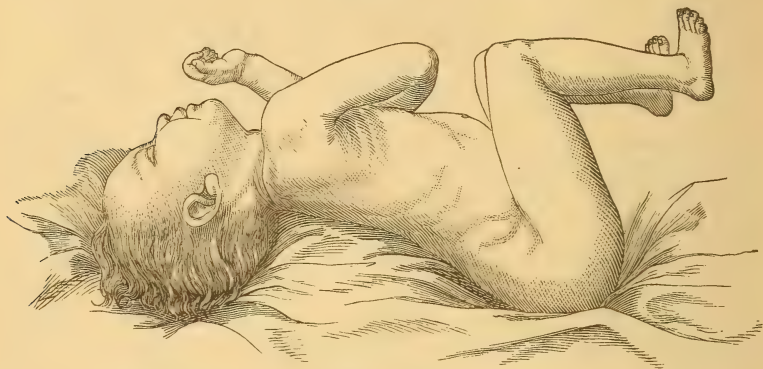
<sup>4</sup> Essays on Fevers and other Medical Subjects, Middleton, Conn., 1823.

<sup>5</sup> Aitken's Science and Pract. of Medicine, pp. 492-505, 3d Amer. edit.

<sup>6</sup> Med. and Surg. Memoirs, pp. 412-507.

*The head is drawn backwards and downwards, and the muscles at the back of the neck are rigidly contracted. When the head is forced forward, or when the child bends forward to drink, the pain is greatly aggravated. At the same time the pupils are contracted. The child moans constantly, and is restless; this is an early symptom, and may appear at the end of twenty-four hours, and be the first to attract our attention.*

Fig. 59.



(J. Lewis Smith.)

The pulse is now quite rapid, and may beat 100 to 120 per minute. The pain meanwhile increases, and affects the head as well as the entire length of the spine, and is increased by pressure. Just as in other forms of meningitis, the movements made by the patient aggravate his suffering, and he usually strives to keep quiet. He is conscious for the first two or three days should he live so long, but at the end of this time he loses his intelligence after first growing delirious. The pulse, temperature, and respiration are increased. The former sometimes beats 130 per minute, while the thermometer may indicate an advance of  $104^{\circ}$ , but it usually remains at about  $100^{\circ}$ . At an early period crops of herpes appear upon the face and limbs, and the skin is hyperæsthetic, and the patient cannot bear handling. After the first ninety-six hours the convulsions succeed the primary rigidity. Opisthotonos or other tetanic contractions make their appearance. Stupor follows, and he dies in a condition of coma; and according to Niemeyer death takes place with symptoms of œdema of the lungs. The bowels are constipated during the entire disease, and during the later stages the patient has involuntary discharges of urine.

The above description is of an ordinary case. There are great variations, and either death may take place in a few hours, or there may be a tardy convalescence accompanied by structural changes of a very serious nature. The course of the disease may open with chill followed by rapid convulsions and coma, when the patient may die in less than twenty-four hours.

In other cases, after the subsidence of the acute symptoms, which may last for a week or two, convalescence takes place, attended by headache

and muscular contractions, which continue for some time. Deafness very often results; and I have several times met with total loss of vision, and paralysis of some of the facial muscles. In one case brought to me from the interior of the State, there was rigid contraction of the muscles at the back of the neck; and in another, seen with Dr. F. H. Rankin, now of Newport, besides ptosis, and paralysis of the pharynx, there was an otorrhœa with extensive middle-ear disease. This patient was quite an imbecile, intellectual impairment having begun after the subsidence of the acute stages. One of these chronic cases has been under observation for several years, but I have been unable to effect more than trifling improvement.

**Causes.**—Epidemic cerebro-spinal meningitis seems to be much more common during cold weather, and is much oftener met with during infancy than at any other period of life. Adults are not exempt; but the disease prefers the young. It is a disease, like typhus, which usually attacks the poor; and bad ventilation and insufficient food seem to prepare the way for epidemics. In the city of New York the first outbreak of the disease appeared in 1866; and subsided, to reappear, February, 1872. In the sparsely settled wards of the city (the 19th, 20th, 22d), where building was going on and fresh earth turned up, it seemed to prevail. There were 45 fatal cases during the winter quarter in these wards, while the entire number of deaths in New York during the same period from this cause was 108. During the spring quarter there were 492 deaths, 148 being in these wards. It subsided in the spring of 1873, but reappeared during the autumn of that year. It would seem, from these statistics, that overcrowding had but little to do with the disease, but that bad drainage (this portion of the city being imperfectly drained) had undoubtedly some influence.

**Morbid Anatomy.**—The meninges of the brain show evidences of intense hyperæmia, the sinuses being distended with blood which slowly coagulates, and the dura mater is the seat of ecchymotic spots. There is usually a sero-purulent exudation beneath the arachnoid, and this is found at the base of the brain as well as in the ventricles. It may be recognized, also, in the different fissures and sulci. The spinal meninges are the seat of the same exudation, it being found beneath the dura, or between the arachnoid and the pia mater. All of the spinal membranes are vascular, and opaque in spots. The exudation appears to be confined to the posterior parts of the cord; and usually, when infiltration in the cord has taken place, small elevations may be observed beneath the pia mater. According to the German pathologists, the cervical portion of the pia mater is not commonly the seat of exudation. The membranes are often adherent, and patches of false membrane are visible, so that sometimes the sub-cerebral nerve-trunks are bound together and connected by bridges of organized lymph. The nervous tissue proper is extensively softened in rare cases—especially if the inflammatory action has been at all severe. Spots of localized softening are, however, not uncommonly observed.

**Diagnosis.**—Cerebro-spinal meningitis sometimes resembles certain



irregular forms of malignant malarial fever, on account of intermissions in the febrile state. This is the case more especially during convalescence, when the affection assumes a periodical character. The chill in cerebro-spinal meningitis is not so marked as in the true malarial affection, and contractions of the muscles are rare in any form of malarial trouble. The other points of difference may be thus summed up:—

## CEREBRO-SPINAL MENINGITIS.

Bowels constipated.  
Pulse and temperature do not suffer rapid variations.  
Temperature does not undergo periodical changes.  
Face flushed; eruption.  
Delirium and coma not affected by large doses of quinine.  
Increase of fibrin, and rapid coagulation of blood when drawn.

## CONGESTIVE PERNICIOUS MALARIAL FEVER.

Not usually so.  
Both subject to great variations, feeble and irregular (Jones).  
Temperature undergoes decided periodical changes.  
Complexion sallow.  
All symptoms modified usually by negative treatment with quinine.

A malignant typhus, or a masked variola, might counterfeit cerebro-spinal meningitis; or, on the other hand, acro-narcotic poisoning might simulate the affection. The presence of tetanic spasms of the post-cervical muscles is, however, so prominent a symptom that when it is absent the improbability of cerebro-spinal meningitis is considerable.

**Prognosis.**—This disease, like other forms of meningitis, has a bad character. Death is generally the rule, recovery the exception. In the city of New York the total number of deaths from all causes was 29,084 during the twelve months ending Dec. 31, 1873. Of these, 9593 were placed under the head of zymotic diseases; and the number of deaths due to cerebro-spinal meningitis was 290. Of these, 69 were under one year, and 164 under five years. Very few cases were over thirty. In the majority of cases the disease runs its course in from 4 to 20 days. In fatal cases death occurs generally before the 12th day.

**Treatment.**—In regard to treatment, little can be said that will be encouraging. The ordinary antiphlogistic treatment, consisting of abstraction of the blood by leeches applied to the mastoid processes, and bladders of ice to the head, and large doses of calomel, according to some observers, have cut short the disease, especially when these remedies were used at its commencement. The almost wonderful results that have followed the use of ergot in large doses suggest this remedy to us, and I have no doubt that it will prove to be very efficacious. Ziemssen recommends morphine, and has never observed any unpleasant effects following its employment.

## CEREBRO-SPINAL SCLEROSIS.

**Synonyms.**—Sclérose en plaques disséminées (Charcot and Bourneville); Insular sclerosis (Moxon).

**Definition.**—A disease of the human system, the essential lesions of which are patches of neuralgic degeneration irregularly scattered through

the nervous substances of the brain and spinal cord, and involving chiefly the motor tracts.

For a long time this disease was mistaken for paralysis agitans (Parkinson's disease), chorea, and other neuroses; and even after it had been shown to be a separate neurosis a certain amount of confusion existed in regard to its nomenclature and its position among the scleroses. Charcot and Moxon<sup>1</sup> were the first to give it a distinct character.

**Symptoms.**—We may divide the progress of the disease into three stages.

*1st Stage.*—The first symptom, which is common to several other neuroses, is gradual loss of power in the lower limbs, which, by itself, does not attract attention to the grave nature of the disease in its incipency. With the weakness there is no atrophy and no loss of sensation, while reflex excitability is either normal or only slightly increased. The rectum is not affected, nor is the bladder, and there is simply a paresis which lasts for a variable time, perhaps for two or three months, or for a much longer period. The partially paralyzed limbs become agitated by tremors, which are seen best when the patient takes some constrained position, or attempts to walk a straight line. He may have the gait of an ataxic, but generally the walk is more like that of a general paralytic, being characterized by weakness of the extremities. As the disease invades a higher portion of the cord, we will find tremor of the upper limbs and paralysis of the cranial nerves, indicated by symptoms I shall describe in speaking of the descending variety. I may allude, however, to a particular defect in articulation, the patient being unable to pronounce some of the labial consonants.

*2d Stage.*—Rigidity of the limbs supervenes, with various contractures of a spasmodic character, and exaggeration of the tremor. One of my patients died in her bed with her knees drawn up to her chin, her legs flexed on the thighs, and her arms drawn closely to her chest. It required quite violent exertion for me to extend the limbs, and the tremor was markedly aggravated when I did so. Electro-muscular irritability is next greatly increased, and reflex excitability heightened. Epileptiform attack may now appear, as well as apoplectiform, and death may occur at this period from the invasion of some cerebral vessel and consequent cerebral hemorrhage.

*3d Stage.*—This stage is marked by rapid decline of the patient's strength. Incontinence of urine and feces, bedsores, and dementia follow, and, after other evidences of gradual wasting away, death may end the scene.

The course of this form is: *First*, paresis of lower extremities and tremor; *second*, contraction, and aggravation of tremor; *third*, general dissolution.

**1st Stage of Descending Form:** This is the condition of affairs when

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<sup>1</sup> Eight cases of insular sclerosis of the brain and spinal cord, by W. Moxon, M. D., Guy's Hospital Reports, vol. xx., 1875.

the cord is attacked *secondarily*. When the disease begins in the brain, the early symptoms may be headache, convulsions, vertigo, or, what is more common, paralysis of some of the cranial nerves; there may be ptosis, strabismus, loss of hearing, and facial paralysis, or troubles of speech and embarrassment in swallowing. The important symptom next in advance is the appearance of tremor, which is first seen in the tongue, which, when protruded, trembles visibly; or it may affect the lips, as may be noticed when the patient speaks. The eyeballs oscillate (nystagmus), and the head may become agitated, and afterwards the upper extremities. A peculiarity characteristic of all forms of sclerosis is not absent here, viz., the aggravation of tremor by voluntary efforts made to control it, and its diminution during rest. If the individual attempts any complex action, he is utterly unable to complete it properly, for the movements increase until muscular control is entirely lost. I have alluded to the lost sense of location, which is also seen in advanced locomotor ataxia, and I may state that it is also a symptom of this form of sclerosis.

2d Stage: The limbs lose their power to a great extent as the disease advances, and permanent contractures of the upper and lower limbs, which by this time are affected, render the patient very uncomfortable. His forearms may be flexed, and the fingers are doubled up, as is the case in uncomplicated lateral sclerosis. The thighs are even flexed on the pelvis, and the legs may be as well. The knees are approximated quite forcibly, and it is often difficult to separate them. This stage may last for several years.

3d Stage: Meanwhile the tremor has continued, and increased in violence; but it may sometimes be stopped by flexing the great toe, just as Brown-Séquard has shown may be done in epilepsy. The bladder and rectum are now involved, and the patient suffers terribly from cystitis, and is prostrated by diarrhoea. Bedsores form, and he gradually sinks into a state which invariably has a fatal termination. In both varieties there is great difficulty in articulation, and disturbance of function in those organs supplied by the lower cranial nerves. The lower lip falls, and there is dribbling of saliva, while food often remains in the mouth wedged between the teeth and between the gums and cheek, and liquids find their way through the nostrils. Beyond slight irritability and restlessness, there are usually no mental symptoms at the outset, or until the fixed stage, when sometimes there is intellectual as well as physical decay; but this is not the rule. A case which seems to be of great interest, because of the atrophy of the upper limbs, came under my notice two years ago.

E. W., aged 37, salesman, no family history of nervous trouble. Father and mother alive; nothing to account for his present condition. Five years ago he was employed in a drygoods store, and his attention was called to a slight weakness in his thumb and forefinger of the right hand when he used his scissors. There was subsequent tremor, which annoyed him excessively, and which subsequently became quite general. About the same time he was subject to very severe headache, vertigo, and some-



times vomiting. The tremor meanwhile increased, and it became so violent when he attempted to execute some fatiguing act that he was forced to desist. He next noticed that his vision was beginning to be impaired, that he saw double, or that "mist floated before his eyes." The trembling continued, and when he came to me I found his condition to be as follows: The patient is a tall man, of decidedly nervous temperament, quite feeble and emaciated, and very much depressed. Both arms are convulsed by tremors, but especially the right. The biceps and the extensors of the hand are much atrophied, and there is great loss of power. He tells me that the tremor has been much more violent than it is now. The sensibility of the cutaneous surface is rather lowered, and there is a certain amount of analgesia, so that pins may be run into the dorsal aspect of the forearm without producing pain. He was able to press the fluid in the dynamometer up to 7.50 with the right, and to 17 with the left. There is still headache at times, and some dizziness. The left eyelid seems to cover the eyeball more fully than the right, and the muscles of the left side of the face were trembling quite violently. When I told him to whistle, his lips trembled so much that he could not do so; and when I requested him to repeat the line "Ben Battle was a soldier bold," he did it as follows: "Me-e-n m-m-m-etta was a s o o g a m-mold." His articulation was quite defective, and I had great difficulty in understanding him. His tongue trembled, and his lower lip seemed to sag and fall forwards, and he was obliged to wipe his mouth quite constantly, as there was a considerable escape of saliva. When I told him to hold his head in such a position that I might examine his eye with the ophthalmoscope, it shook to a great degree, and I had difficulty in illuminating the retina. He says this is recent, and that his head was not affected by tremor until a month or two ago. His mind is clear, and his memory unimpaired. I have seen him but once, and there has been no advance in his condition.

The following case is reported by Bourneville:—<sup>1</sup>

Rosine Spitale, 20 years old. At 17 years of age she was suddenly affected (after crossing a small stream and becoming chilled) with loss of power, first in the right lower extremity, and then in the left, and some time after the hands began to tremble. At 18 there was some subsequent improvement, but it was very slight. Soon afterwards menstruation ceased, and some time after this the symptoms reappeared. Hemiplegia occurred without loss of consciousness or convulsions, and the tongue and eyes were involved. The disturbances of sensation were moderate; there was a certain amount of numbness in the lower limbs, and a sense of clumsiness of the tongue, with difficulty in articulation, and some diminution of mental power. At the beginning of 1853 the patient was well nourished. A half grain of strychnine daily has produced an amendment for ten or twelve days. Electrization produced movements in the lower limbs, and increased the trembling in the upper extremities. In the course of the month the paresis of the inferior extremities was nearly complete, the trembling of the eyes with dilatation of the pupils is quite pronounced, and the patient has become very stupid.

\* The intonation was very much like what we would expect to find in "cleft palate."

<sup>1</sup> La Sclérose, etc., Paris, 1869, p. 92.

*January, 1854.* The hands tremble less than they did. There are involuntary discharges of urine. Ergot 3ij per day has been used for several months. It acted once upon the sphincters, and seemed to improve the weakness of the limbs, for several movements were possible.

*Spring, 1854.* Bedsore on sacrum.

*September.* In a state of decline; the bedsore has extended very rapidly; pain in the head; pulse 136.

*October.* Repeated rigors; sensibility of the inferior limbs returned; feebleness of the extensors of the back; scoliosis toward the right; the trembling in the extremities persists.

*November 1.* Death, preceded by involvement of the muscles of the pharynx.

*Autopsy.*—The gray matter is hard; the nervous substance in the neighborhood of the lateral ventricles and that of the protuberance were hard. We found gray nodules superficial and deep. The white substance had become hard in spots. Beneath the microscope the indurated nodules (white) consisted of a fibrous, moss-like, connective tissue; the elements of the nervous matter had almost entirely disappeared; and the white nodules were pressed beneath the surface of the cut. The spinal cord was indurated. The great vessels and viscera were healthy.

Dr. Geo. S. Gerhard<sup>1</sup> has presented the following interesting case of this disease:—

Samuel A., æt. 57, a native of Ireland, and a blacksmith by trade, was admitted into the out-patient department of the Infirmary for Nervous Diseases on September 17, 1876, and gave the following history. His health had always been good until about seven years ago, when, after no known cause, he began to lose power in the legs. One year after this his arms grew weak, and he then observed for the first time that any movement of the upper or lower extremities was accompanied by tremor. At a somewhat later period his speech became affected. The weakness of his limbs and the trembling gradually increased, until finally, about four years ago, he was obliged to give up work.

On admission there is decided loss of power in the upper and lower extremities, and upon his attempting to use either, a large and jerky tremor is developed. He walks with the assistance of a cane, but his movements are slow, and his feet clear the ground with much difficulty. His grip; particularly that of the right hand, is feeble, squeezing the dynamometer with the former to 100° and with the latter to 110°. In the upper extremities the trembling is especially well shown during the performance of an act requiring some little time for its execution, such as lifting a glass of water to the mouth. The tremor also involves the muscles of the head and trunk, but it ceases entirely when the patient is in a state of absolute repose. There is no muscular wasting, no loss of electrical response, and no disturbance of sensibility.

His mental faculties are decidedly impaired, and his speech is thick and deliberate, there being a decided interval between each word. His eyesight is poor, and examination of the fundus reveals commencing atrophic changes, as shown by attenuation of the vessels and a general pallor of the optic disk; there is also slight nystagmus. The unsteadiness of gait and the tremor are not increased by closure of the eyes. His urine

<sup>1</sup> Philadelphia Medical Times, November 11, 1876.

is in all respects normal, and he has no loss of control over the bladder or bowels.

**Causes.**—Jaccoud is of the opinion that sclerosis occurs as a disease of childhood, or adult life up to 45 years, and that there is nothing to indicate the special liability of either sex; whilst Charcot considers it a disease which is much more common among females than males, and that it rarely appears after 40. Of six cases I have recorded their respective ages were 18, 26, 33, 37, 41, 46; four were males and two females. Of eighteen cases collected by Bourneville fifteen were women and three men. In three of these the disease began between 36 and 40, three between 30 and 35, and the others between 15 and 30. Very little is known in regard to the etiology of sclerosis; but "moist cold," emotional excitement, and venereal excesses are spoken of by the different Continental writers as causes.

Bourneville found that the greater number of his cases died between 35 and 50, and that the disease appeared in most instances between the ages of 26 and 35. In one of my patients the disease began at the 5th year, in another at about the 18th year, and in the third and fourth at 32, and in the fifth and sixth between 35 and 40.

**Morbid Anatomy and Pathology.**—I have spoken in another chapter about the morbid appearances in sclerosis, and nothing remains to be said in regard to this particular form. It is only a question of location that concerns us, and after death we will probably find patches of tissue scattered through the brain and cord. The antero-lateral columns seem to be invaded in nearly all cases, and this would appear probable from the contractures.

**Diagnosis.**—In the *ascending* form it must be remembered that the tremor follows the paresis, while the descending form is characterized by tremor as a primary affection, or at least before the muscular paresis of the extremities. Paralysis agitans may be confounded with the descending form of the advanced disease; the tremor in the former disease is continuous, and is often not affected by quieting influence or sleep, but is not aggravated by efforts of the will. The early symptoms of this form may also point to progressive paralysis of the insane, and to intracranial tumors; but the subsequent progress of the affection, the development of new symptoms, and the common absence of neuro-retinitis, are sufficient to remove any doubts as to its true nature.

**Prognosis.**—Invariably bad.

**Treatment.**—I know of no remedy that can reconstruct a degeneration of nerve-tissue which consists in proliferation of connective-tissue cells, and nerve-tube disappearance. Nitrate of silver, tribasic phosphate of silver, chloride of gold, galvanism, bichloride of mercury, and chloride of barium have been all used. It seems that only one chance may exist—the possibility of syphilis. If this be present, it is probable that specific treatment will be successful. We are to improve the patient's general condition, and relieve his tremor either by conium or hyoscyamia, and make him as comfortable as possible.



## ALCOHOLISM.

## ACUTE—CHRONIC.

**Synonyms.**—Ebrietas, Alcoholismus, Delirium tremens; Mania à potu, Alcoolisme; Trunksacht; Chronic alcoholic intoxication (Reynolds).

**Definition.**—A disease of the nervous system resulting either through direct action of alcohol upon its tissues, or through impairment of other organs which fail to remove effete substances from the blood; and symptomatized by mental aberration, and by various sensorial and motorial phenomena, usually the result of lowered functional activity.

The immoderate use of alcoholic beverages is usually followed by the most deplorable consequences. Sad to say, this condition is too familiar to need any extended description, as far as the appearance of the patient is concerned; but there are other features of the disease that need earnest and careful study.

The effects of alcohol upon the human being may be said to be physiological and pathological. The sensorial alterations are much more interesting than the motorial, and of these we will speak in detail.

The imbibition of a moderate amount of alcohol, as we know, is usually followed by a general feeling of comfort, a certain degree of exhilaration. The individual is no longer absorbed in himself. He is animated and gay, his ideas flow rapidly, and he becomes filled with greater energy and endurance. If the dose be increased, the mental functions become more active. He is excited and demonstrative, and either violent and noisy, or tender and maudlin, according to the thoughts which have most engrossed his attention, or through the influence of temperament. Incoherence of speech and confusion of ideas succeed the ordinary mental excitement, and this may be followed by a condition of stupor, the individual becoming perfectly unconscious of injury, and unmindful of either bruises or cuts, or even severe burns. He may stagger and fall, and lie in some exposed place regardless of the blaze of the sun, the flies, and the noise. He has finally become reduced to what Magnan<sup>1</sup> calls “la vie végétative.” He is “dead drunk.” This deep alcoholic stupor may last for some time, and end the patient’s career; or he may become maniacal instead, or present the condition described by Percy<sup>2</sup> under the name *ivresse convulsive*, in which, with clonic convulsions, he grows furiously maniacal, grinding his teeth, and cursing and menacing those about him. The maniacal attacks are no doubt influenced to some degree by the character of the illusions and hallucinations.

## ACUTE ALCOHOLISM.

**Symptoms.**—The continued use of alcohol in excess for a week or two, such as occurs during an ordinary debauch, is very apt to lead to an

<sup>1</sup> *Récherches sur les centres nerveux*, p. 116.

<sup>2</sup> “Art. *Ivresse Convulsive*,” *Dictionnaire des Sciences Médicales*, t. xxvi., p. 249.

attack of *delirium tremens*. This state of acute alcoholism may also occur should the patient, who has drunk not necessarily to intoxication, but to a degree almost approaching it, be deprived of his drink.

One of the earliest indications of this state of alcoholism is a tremulousness or "shakiness," which is quite marked in the early part of the day, and is connected with nausea and want of appetite. The patient is restless and irritable, sleeps poorly, and presents an appearance of dejection and sadness. His eyes are red and watery, and his skin is of a muddy color. His features are drawn and haggard, and he is a wretched object indeed. The gastric irritability may be so great as to prevent any retention of food, and the simplest forms of nourishment are ejected by the stomach. Constipation is obstinate, and the urine is passed in small quantities and loaded with the urates, so that a dense brick-dust precipitate is found in the chamber. The attack is immediately preceded by great excitability, and by illusions and hallucinations, which grow very marked as the patient becomes noisy and violent. Magnan has graphically described the different varieties of mental trouble. The patient may be sad and utterly dejected. He may imagine that he has committed some great crime; that he has been sentenced to death; that he is being executed; and these delusions may markedly influence the character of his outward expression. In nearly every case there is some delusion of persecution of a horrible kind. The attack usually begins with hallucinations of a visual character, in which snakes and other reptiles, devils, imps, gnomes, and goblins terrify the patient. In one instance which I remember, he was tortured by devils who held lighted candles, and were about to set his clothes on fire; in another case the patient endeavored to escape a falling weight. The illusions are always followed by hallucinations, and finally by delusions. The irritations of the organs of sense are distorted so that the simplest and most common noises become changed by the patient's disordered imagination into the most terrible sounds. The cry of the vendor in the street is likened to the despairing shriek of a lost soul. The stroke of the clock, a funeral bell, and the voices of those in the room are supposed to be the savage yells of a howling mob. The objects which the patient sees are nearly always transformed into animals, which, controlled by no natural laws, run over the ceiling, or gallop through the air. Odors are reversed, and food is supposed to be poisoned. Animals run over the skin; sometimes they are rats or lizards; and at others he may call attention to the torture inflicted by thousands of needles or cutting instruments. Maniacal outbursts are the common feature of the attack, the patient seeming to possess herculean strength, and it is sometimes necessary to have six or eight strong men to prevent him from throwing himself out of the window, or committing some deed of violence. He may remain in this condition for several days at a time, during which period he neither sleeps nor eats. His eyes are bloodshot, and he sweats profusely. The pulse<sup>1</sup> is very rapid, small, and irritable,

<sup>1</sup> The sphygmograph has been employed by Anstie in cases of *delirium tremens*, and the tracing obtained very closely resembles that of the typhoid fevers and inflammation. It is of a marked dicrotic type.

and though the deep temperature may reach 102° or 103° F., the hands and feet are cold, and the palms and soles clammy.

When recovery takes place, the first change for the better is sleep. The violent symptoms subside gradually in the reverse order of their appearance. He may awake, after fifteen or eighteen hours, irritable, but not much better; or there may be a lesser degree of excitement, more sleep, and gradual improvement.

In other cases death follows, there being a subsidence of the violent delirium, which changes its character and becomes muttering; when he relapses into a typhoid state, and gradually passes away.

The tendency to the commission of deeds of violence is quite characteristic of acute alcoholism. Of 377 cases observed by Bouchereau and Magnan<sup>1</sup> in the year 1870, twenty-four attempted to commit suicide, and nine attempts at homicide were made. These cases were seen under restraint, but among the cases which occur outside of hospitals and asylums, the number is far greater.

Lancereaux has described the features of acute absinthism, which, however, is rare in this country. He agrees with Magnan, that epileptic attacks exactly like those of the ordinary disease follow the immoderate use of absinthe. Several hours after the toxic dose of this liquor has been taken, the convulsions take place, and involve chiefly the muscles of the back and of the posterior part of the neck, so that a species of opisthotonus results. These tonic convulsions are followed by others of a clonic character, affecting chiefly the muscles of the face. There is frothing at the mouth and grinding of the teeth. The muscles of the body are also next in a state of clonic contraction. The actual attack lasts for an hour, and is not followed by coma. It is separated by intervals of comparative quiescence. The patient then falls asleep, and, after a variable time, awakens complaining of sensory disturbances.

In an abstract of Lancereaux's article by Decaisne,<sup>2</sup> an admirable description of acute absinthism is given. He calls attention to the fact that the cry and coma are absent in absinthe epilepsy, and the attack is irregular, and resembles a convulsive attack of a hysterical character.

#### CHRONIC ALCOHOLISM.

**Symptoms.**—A much more grave condition of affairs follows the continued use of large quantities of alcohol, and no more hopeless disease exists than that of which we are about to speak. While in delirium tremens recovery may take place, followed by total reformation, without any serious damage to the nervous system, the more serious nerve-changes wrought by constant saturation can never be repaired, but tend to further degeneration and decay.

Chronic alcoholism begins by a number of insidious alterations in the

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<sup>1</sup> *Op. cit.*, p. 129.

<sup>2</sup> *Revue des Sciences Med.*, No. 33, 1881, p. 231.



nervous substance, whereby its functional activity is embarrassed, and minor symptoms at first, and more grave ones afterwards, appear very gradually and progressively.

The victim of chronic alcoholism may present the symptoms of tremor and loss of power of which I have before spoken. The tremor is rhythmical, and begins at first in the extremities, and afterwards involves the entire body. There seems to be an accompanying want of power, for he relaxes his hold upon any object he may grasp when his attention is diverted. His morning dram involves an effort worthy of a better cause. He grasps the glass with both hands, fearing that he may spill even a single drop of the precious liquid, and carries it carefully to his mouth, clutching the rim of the glass between his teeth, oftentimes with sufficient force to bite out a piece. The lower extremities become involved, and the patient shuffles along in a clumsy manner, his feet being scarcely lifted from the ground. His dress becomes disorderly, and his habits are no longer characterized by neatness and tidiness. His facial muscles lose their play, and his countenance wears a wonderfully woebegone and sorrowful expression. He wanders wretchedly from one grog-shop to another; eats sparingly, and rarely ever, unless his worn-out stomach is stimulated by a dram. He loses flesh, and his clothes hang to his withered limbs like the vestment of a scarecrow. This is but the first step in the advancing disease. Memory becomes weakened, and forgetting even faces and names, he drops one by one his old friends, and sits in loneliness for hours at a time.

The mind is utterly sapped, and he is reduced to a state of dementia. Numerous grave changes occur in addition to these. Speech becomes thick and unintelligible. In the early stages there may be convulsions or attacks of delirium tremens; but one of the most striking and serious expressions of the disease is the occurrence of paralysis; and there may be hemiplegia or paralysis of a local character, the third nerve becoming implicated, and ptosis resulting. The subject of chronic alcoholism is generally anæsthetic, and this to a marked degree. Not only is tactile sensibility impaired, so that he is unable to determine the nature of even a rough object, but he is unaffected by extremes of temperature. In one case which I can recall, this was illustrated by the fact that in sitting before the fire he thrust his foot beneath the grate, and left it there for some time before his position was discovered by a member of the family. Hemi-anæsthesia<sup>1</sup> is spoken of by some writers, but it is an extremely rare feature of the disease, and is probably a late symptom resulting from organic changes on one side of the brain. An anæsthetic condition of the cornea has been alluded to.

Convulsive seizures of different kinds are occasional evidences of the serious effects of alcohol. These may vary from simple spasm to a variety of convulsion which closely resembles a marked epileptic paroxysm. In fact the diagnosis is oftentimes very difficult. What I have said about

<sup>1</sup> Magnan considers that organic hemi-anæsthesia and general paresis are quite common results of chronic alcoholism, *op. cit.*, p. 134.

the mental condition in acute alcoholism may be now applied. The hallucinations and lighter forms of sensory and mental aberration exist at different stages, but towards the end the condition is one of dementia of the most profound character, the patient being completely oblivious of the outside world, and of his duties to society. He is morally irresponsible, and the crimes he may commit are motiveless and dictated only by a diseased mind.

**Causes.**—Chronic alcoholism follows the steady use of large quantities of alcoholic liquors, but is rarely found among those who drink wine or malt liquor. The French, Italians, and Germans are, therefore, seldom affected in their own countries, especially outside of the large cities, where a very small amount of ardent spirits is taken. In England, Scotland, Ireland, and America the case is different, for in these countries there is no low-priced light beverage which takes the place of the wines and beer of the European Continent, which are drunk in preference to water. Without entering into the discussion of the effects of alcohol upon other organs of the body than those of the nervous system, it may be said that the condition known as alcoholism springs from a protracted use of large quantities of strong liquor, so that the nervous substance is deprived of its normal nutrition, the blood being charged with effete substances which should be eliminated by the kidneys, lungs, and skin.

Delirium tremens is due generally to the direct action of a large quantity of alcohol, which produces overwhelming toxic effects; while chronic alcoholism implies a structural degeneration due to the *continued* action of the alcohol itself, and to the vitiated blood.

Delirium tremens may occur either from a sudden cessation of indulgence, or in the midst of a prolonged debauch, most commonly, however, the latter. In some persons elimination goes on so perfectly that large quantities of liquor may be taken and disposed of without any profound effect upon the nervous system being produced. These individuals may drink to a point much beyond moderation, and still suffer no marked inconvenience, the alcohol seemingly affecting some other organ, which may be either the liver or kidneys, so that cirrhosis or degeneration of other kinds may take the place of the cerebral trouble in the beginning.

Males are much more often affected than females, as the statistics of Magnan show:—

|                          |              | M.  | F. |
|--------------------------|--------------|-----|----|
| Acute alcoholism (D. T.) | { 1870 . . . | 35  | 2  |
|                          | { 1871 . . . | 42  | 2  |
| Subacute “               | { 1870 . . . | 216 | 51 |
|                          | { 1871 . . . | 159 | 47 |
| Chronic “                | { 1870 . . . | 126 | 11 |
|                          | { 1871 . . . | 90  | 14 |

This fact has been confirmed by statistics collected by the Health Department of New York. During the year 1873, 45 deaths were reported from delirium tremens, but four of whom were females. It is probable that there were many more cases which were not reported as such.

Women, however, though not so subject to chronic alcoholism as men, often drink to excess, and not rarely develop delirium tremens. This bad habit is confined chiefly to either extreme of society—the very lowest class, or the highest in the social scale. Among the latter the amount of private dram-drinking is astonishing; and though the “skeleton in the closet” is carefully guarded by the friends of the patient, it is by no means uncommon for the physician to be called in to attend cases of delirium tremens in high life.

Absinthe, which is extensively used in Paris, and is beginning to be introduced into this country, produces a terrible form of delirium tremens, in which mania is a marked feature; and a form of epileptiform attack is also quite common.

Alcoholism is much more often observed between the twentieth and the fiftieth year, and is very rare before that time.

As to hereditary predisposition there is a great deal to be said, but when we attempt its consideration we depart from the immediate subject. Occupation and mental influences have much to do with the making of drunkards or hard drinkers. Barkeepers, and individuals exposed to severe weather, are commonly addicted to drink; the one either feeling obliged to be convivial or indulging only because the liquor is so accessible, and the other because he “needs something to keep out the cold.” Mental depression, grief, and business worry are interesting in their social features, but do not strictly come within the scope of an article of this character.

**Morbid Anatomy and Pathology.**—The prolonged use of alcohol is followed by marked changes in the structure of the nervous substance. In the early stages there may be found appearances which are ordinarily met with in uncomplicated cerebral congestion, viz., enlarged vessels, injected meninges, and effusions of serum. These may vary greatly in their extent and appearance, and may be associated with a fatty degeneration of the vascular walls, patches of softening, or even little foci of induration. The disease leaves its traces most indelibly stamped as meningeal thickening and opalescence, and perhaps encysted collections of blood, which have been described in speaking of pachymeningitis. The sinuses are engorged, and the dura mater may be adherent to its underlying membranes; or they, in turn, may be in such close contact in spots with the cortex that their removal necessitates the tearing out of patches of superficial gray substance. The convolutions will be found to be atrophied and reduced in size, and the ganglia at the base are often greatly softened.

Many observers, among them Carlisle and Percy, have found alcohol in the fluids in the ventricles. Besides these intracranial changes, the liver, kidneys, and stomach present appearances with which all pathologists are familiar. The arteries throughout the body are found to have undergone atheromatous degeneration, and this is seen in the brain to a very decided degree. As to the condition alluded to by various observers, viz., the mechanical change exerted directly by the contact of alcohol with the tissues, I think there has been much exaggeration. The sclerosis so often



seen is much more probably the result of interstitial inflammatory change than a chemical transformation.

The experiments made by Anstie,<sup>1</sup> Magnan,<sup>2</sup> Percy, Marcét,<sup>3</sup> and Motét<sup>4</sup> settle with great certainty the pathological processes which follow the toxic administration of alcohol. Anstie took a full-grown dog weighing 10 lb. 4 ozs., and injected 6 ozs. of mixed alcohol and water into the stomach at 1 P. M. No food had been taken for four hours previously.

1.4 P. M. Animal obviously affected; staggers in walking, and frequently falls down. The hind quarters are weak, and skin of hind limbs insensitive. Resp. 24; circulation, 140.

1.6 P. M. Dog lies extended on the floor quite drowsy, but capable of being roused; fore-limbs retain slight degree of voluntary power. Tongue protruded, and the dog "slavers" still. Skin about mouth anæsthetic; conjunctiva sensitive.

1.7.30 P. M. Animal falls on its side, comatose and snoring. Conjunctiva insensitive with other parts. Resp. 20; circulation, 184, tolerably strong. Ano-genital region was sensitive to painful impressions. Pupil strongly contracted at first, but became dilated at 1.25, little sensitive to light; anæsthesia remained; eyes still insensitive; continuous tremor of hind-legs began and continued for a short time. Respiration declined in frequency, and became gasping, and ceased at 3.5 P. M., two hours after the ingestion of the alcohol, the heart beating 64 per minute. It remained irritable for some minutes later. Much more complete and earlier coma followed the administration of larger doses.

The continued toxic use of alcohol produces changes not only upon the nervous system directly, but secondarily through other organs which are primarily affected. A large quantity of alcohol taken into the system induces pathological changes somewhat after the following manner: A certain portion, quite small in amount, is promptly excreted, and may be detected in the breath, urine, bile, and sweat, while the greater proportion remains in the blood, greatly altering its character and inducing a large number of interesting changes. Lallemand, Marcét, and various experimenters have found that the excretions contained much pure alcohol, and others have detected, by the chromic acid test, traces of alcohol forty-eight hours afterwards. Anstie declares, however, that but the *merest fraction* of the amount taken is eliminated in its unchanged form. In this conclusion he differs from the authorities I have quoted. The alcohol remaining in the blood is partially eliminated in its decomposed state (carbonic oxide and water), while a certain quantity remains. The internal organs are congested, notably the liver, kidneys, and lungs, so that excretion is very slowly performed, and the urine voided is scanty in amount, devoid of the chlorides, and rich in urates. The blood circulates sluggishly, and

<sup>1</sup> Stimulants and Narcotics, p. 335 et seq.

<sup>2</sup> Op. cit., p. 116.

<sup>3</sup> De la folie causée par l'abus des boissons alcooliques, thèse de Paris, 1847.

<sup>4</sup> Considérations générales sur l'alcoolisme, et plus particulièrement des effets toxiques sur l'homme par la liqueur d'absinthe, 1859.

contains fat and sugar. I have also found sugar in the urine, which probably resulted from irritation of the medulla as well as certain disturbances of kidney and liver function.

The abundance of carbonic acid requires double duty upon the part of the lungs, and consequently respiration becomes labored and quickened. The natural oxidation of the blood is seriously embarrassed, and elimination is retarded most seriously.

The nervous system of course suffers from this change in its badly nourished state. Degeneration of the nervous elements follows, and interstitial thickening and medullary metamorphoses take place, so that the loss of function is very great. The pneumogastric being implicated, the lungs and other organs are not properly innervated, and many of the curious evidences of such disorder follow. This is illustrated by the tendency to pneumonia which often exists as a feature of alcoholism.

The sympathetic system is of course implicated. The actual presence of alcohol is attended by vaso-motor paresis, and a number of vascular changes probably follow. It might be well, before closing, to refer to a condition of the cranial bones noted by Lancereaux and others. A hardening and thickening is due to nutritive changes, which Anstie thinks is not a true hypertrophy, as the original texture of the bone is lost.

**Prognosis.**—A table prepared by Mr. Neilson from the Registrar-General's report shows that the probable duration of life in individuals who have reached the 20th, 30th, 40th, 50th, and 60th years, and who have been either temperate or intemperate, is about the following:—

| Having reached the age of | Has an average chance of still surviving | But the intemperate have an average chance of surviving only                  |
|---------------------------|--|---|
| 20                        | 44.21 years                              | 15.53 years, or 35 per ct. of the duration of life of the general population. |
| 30                        | 36.48 "                                  | 13.80 " " 38 " " "  |
| 40                        | 28.70 "                                  | 11.62 " " 40 " " "  |
| 50                        | 21.25 "                                  | 10.86 " " 51 " " "  |
| 60                        | 14.28 "                                  | 8.94 " " 63 " " "   |

This applies only in a general way to the subject, but is significant in showing how greatly the alcoholic habit diminishes the patient's chances. In regard to the prognosis of the actual attack, there is rarely any reason to fear a fatal termination unless the patient has had a number of previous ones. Coma and convulsions should be looked upon with grave suspicion, as they greatly diminish the patient's tendency to recovery. Chronic alcoholism is more unfavorable. Should the patient survive his immediate nervous trouble, it is very likely that disease of some other organ will carry him off. Cirrhosis is the most common of these, and the patient's mental condition may be for some time aggravated by cholesteraemia. Much depends upon his ability to reform; and no assurance can be given that he will recover until this is accomplished.

**Diagnosis.**—The only diseases for which alcoholism may be mistaken

are: 1. General paresis; 2. Sclerosis, and paralysis agitans; 3. Softening; 4. Dementia.

1. General paralysis differs from delirium tremens in the fact that in the former the delusions are always pleasurable and exalted. The general paralytic is the king, the capitalist, the ruler of the universe; the alcoholic patient is depressed, dejected, and sad. These differences, taken into consideration with the fact that the patient suffers from anorexia, that his face is flushed, and the conjunctivæ red, ought to settle the real nature of the trouble. Anstie<sup>1</sup> alludes to the presence of acne as a pathognomonic sign. Chronic alcoholism may very closely resemble general paresis, but there is more proper dementia in the latter.

2. Sclerosis and paralysis agitans are sometimes confounded with chronic alcoholism when there is much disturbance of co-ordination. The tremor and inco-ordination are much greater during voluntary action, however, in the first conditions, and there is rarely any mental disturbance in either.

3. Softening resembles chronic alcoholism, but the paralysis and speech disturbance are much more pronounced, there generally being aphasia, and the headache besides is quite different from that of alcoholism.

4. Senile dementia may make the diagnosis somewhat difficult. The previous history of the patient, however, will generally clear away any doubts that may arise.

**Treatment.**—The physician's first attempt should be to prevent the patient from further indulging his depraved appetite. How this is to be accomplished depends very much upon his surroundings, temperament, and condition. If the attack arises during a debauch, I prefer to cut off *at once* the supply of alcohol, unless he is utterly prostrated. If the attack occurs after cessation, we may then give small quantities of stimulants, and "taper off." Should he be irritable and excited, immediate recourse to sedatives and hypnotics should be had. I have great faith in the bromides, lupulin, or simple remedies of this class. Fifteen or twenty grains of the bromide of calcium, given in a drachm of the tr. lupulin twice or three times a day, is often sufficient to quiet the nervous state. A good cathartic which shall increase the action of the liver, and hasten elimination of the alcohol, is an early form of treatment which is generally recommended. Should the insomnia be troublesome or the delirium violent, we may administer either the bromides, or the mono-bromide of camphor, which I make the claim of being the first to use for this purpose. It may be given in pilular form, made up with confection of roses, in doses of five grains every hour until sleep is produced. The bromides of calcium or sodium in thirty grain doses every two hours sometimes succeed, or, better still, they may be combined with chloral hydrate, so that the patient shall take fifteen grains of each every two hours until the excitement subsides. Cannabis indica has enjoyed great

<sup>1</sup> Article on Alcoholism, Reynolds's System, American Edition, vol. i. p. 677.



popularity in the treatment of this trouble, and should be given in doses of from one-half to one grain of the extract. Should the maniacal excitement be intense, I know of no better remedy than morphine administered hypodermically, but *not by the mouth*, as it may lie unabsorbed for some time with producing any effect; and the physician may be tempted to give still more than the ordinary dose, when to his surprise absorption takes place, and its cumulative action follows. Digitalis has been recommended in large doses, and Anstie preferred the powder because the alcohol of the tincture interfered with the proper action of the drug. I am inclined to think that the application of digitalis stupes to the lumbar region and the abdomen favors kidney action, and does more good than when the medicine is given by the mouth.

It is of importance that the action of the skin and bowels should be increased. For the first object, small doses of tartar emetic assist the emunctory action of the skin, while the compound jalap powder induces copious and watery discharges from the bowels. Cold to the head, either by ice-bags or cloths wet with ice-water, blisters to the calves, and local abstraction of blood may be resorted to in violent cases. As to food: when the worn-out stomach refuses all ordinary articles of diet, it will rarely reject iced milk, which may be given in all cases. After a while soups, nutritious broths, or bouillon made from beef, or Valentine's beef juice, or Borden's extract of beef, either of which is preferable to the Liebig extract on account of the nauseous taste of the latter, may be given in liberal quantities. Small doses of carbonic acid, seltzer, or Apollinaris water, or coffee may be administered before eating, and gently stimulate the stomach, in this respect taking the place of the drams.

The patient's nausea may be corrected by the aromatic spirits of ammonia, or bismuth and morphine, the latter in very small doses.

In chronic alcoholism the aim of the physician should be to restore the normal action of the viscera; to stop the supply of drink; and to freely administer the various preparations of iron, quinine, and phosphoric acid, as well as cod-liver oil. I have found that dialyzed iron is well borne by the irritable stomach, does not constipate, and is therefore an excellent remedy. This may be given with tr. digitalis and tr. nux vomica.

## NICOTINISM.

When the nervous system is subjected to the influence of tobacco in excessive quantities a train of symptoms may be manifested indicating a condition of affairs that may ultimately assume a serious character. While I believe tobacco to be one of the most valuable articles of comfort we possess, I every day am made aware that in an insidious way it produces nervous disorders which are sometimes quite as formidable as those caused by alcohol. I have found in more than one case of general paresis that the immoderate use of tobacco, had, in those of unstable nervous temperament, all to do with the development of the disease. I have no intention, however, of entering into the discussion of its general bearings in relation to public health and the morals of the community, for these sub-

jects have been frequently discussed by popular reformers—and not always temperately or truthfully—but I will briefly call attention to the nervous expression of chronic tobacco poisoning.

**Symptoms.**—The question of tolerance, in connection with physical development; the effect of the constant use of tobacco upon the nervous individual—the possessor of the insane neurosis, perhaps—enter largely into the genesis of nervous symptoms.

In persons of full habit, of phlegmatic temperament, and fat-making tendency tobacco may be used in considerable quantities and quite constantly without other than trifling effect, and in the rheumatic diathesis it is positively beneficial. In the spare, nervous individual the case is different, and the careless and continuous use of tobacco often produces a train of motorial and sensorial symptoms of varying grades of gravity. Both the voluntary and involuntary muscles may be affected, and atonic action of the unstriated muscular fibre result in a variety of cardiac and digestive disturbances.

The action upon the heart is decided, there being great feebleness and inequality of the pulse, and as the brain becomes the seat of chronic anæmia we find dizziness, headache and melancholia, besides a variety of light mental troubles. The muscular tissue of the stomach, intestines and lower bowel are enfeebled so that slow digestion and loose evacuations are consequent.

The production of general muscular weakness is a very conspicuous manifestation of the depressed tone of the nerve centres. These may be expressed either in tremor, slight paresis, or an epileptoid condition; the tremor, however, is the most familiar of all disorders of motility.

It may be unilateral, but is usually found on both sides, the upper extremities being more often its seat than the lower, and like the same motorial disorder seen in alcoholism, and among opium eaters it may be overcome for the time by recourse to the cause. It is essentially the tremor of debility, and has no very regular character. If the smoker extends his hand so that it is in a somewhat constrained position, he will notice that some fingers are more agitated than others, notably the second and third.

An advanced grade of motor feebleness is expressed in paresis, but rarely by paralysis, so far as complete and diffused anæsthesia is concerned. Erb, under the head of toxic spinal paralysis, speaks of the influence of tobacco in its production, and says that it causes lasting paralysis when the toxic action is slow and repeated, and much more rapidly than when acute. There is usually diminution of electro-muscular contractility. Various other disorders of motility are shown in local spasms, and among them are painless facial twitchings and blepharospasm, which may be very distressing; spasms of the limbs and starting during sleep. Not a small number of cases of chronic tobacco poisoning, as I have said, end in the direct production of serious organic disease of the brain, and symptoms in many respects similar to those of cerebral softening or general paresis will be expressed. The pupil presents no

constant appearance that may be considered important. Some authors, among them Taylor, and Woodman and Tidy hold that it is dilated in acute poisoning, while Pereira and Bartholow, say contracted, but in chronic nicotinism it is usually dilated. The urine is copious and loaded with earthy phosphates. Various dysæsthesia are common in chronic nicotinism. The patient calls attention to tinnitus, "tightness about the throat," "pains beneath the ears," as well as intercostal pains, coldness of the feet, crawling sensations, and a sense of feebleness, especially in the morning.

Amaurosis is one of the indications of anæsthesia. <sup>1</sup>Drysdale reported the cases of two young men who became amaurotic from the continued use of tobacco, in one case the man taking but half an ounce of tobacco a day. <sup>2</sup>Masselon in an admirable thesis refers to the production of color blindness, one of his patients being unable to tell a piece of silver from a piece of gold, and in all cases the patients seemed to lose the faculty of distinguishing yellow and red from other colors.

<sup>3</sup>Webster, in a very careful paper, has called attention to the amblyopia produced by tobacco, and fully believes that tobacco *alone* may give rise to this ocular trouble. In seven out of twenty cases he found incipient atrophy of the optic nerve. In 18 of Webster's cases alcohol and tobacco were used to excess, and in one case tobacco was used excessively from ten to fifteen years, and alcohol moderately, and an occasional glass of gin was taken. In one case in which the amblyopia seemed to be wholly due to the abuse of tobacco, the vision rose from  $\frac{5}{200}$  to 70 in each eye when the patient abstained from its use, and received appropriate treatment. Dr. Ely takes a more conservative view of tobacco poisoning as a cause of amblyopia.

Cutaneous hyperæsthesia or anæsthesia are by no means rare symptoms of chronic tobacco poisoning. I have in patients repeatedly found anæsthesia of the lips and tongue, and in one subject smell was abolished, and not restored until the patient was subjected to a course of strychnia. Tactile sense is sometimes blunted, and especially is such the case in the tips of the fingers. Neuralgic pains are by no means uncommon, and are perhaps among the early sensory troubles. These pains may counterfeit those of early locomotor ataxia, and create great misery. In other cases there may be cardiac neuralgia, resembling in many respects the pain of angina pectoris. So grave is this symptom that even medical men who smoke to excess often believe themselves to be the subjects of this affection. Vague muscular pains, shortness of heart, and fatigue after slight exertion all come in for a share of our attention.

The mental expressions of nicotinism are exceedingly variable, and may consist in the beginning simply of a change in the temper and disposition, evinced by irritability, and accompanied by loss of memory, irresolution and hypochondriasis; or in a graver form we may find actual

<sup>1</sup> British Medical Journal, Sept. 5, 1874.

<sup>2</sup> Thèse de Paris, 1872.

<sup>3</sup> Medical Record, Dec. 11, 1880.



symptoms of insanity, illusions, hallucinations and delusions either insane or otherwise, attacks of extreme excitement amounting to mania, or perhaps mania itself.

<sup>1</sup>Bucknill and Tuke speak of tobacco poisoning in the causation of insanity, and <sup>2</sup>Kirkbride reported four cases of insanity due to tobacco. <sup>3</sup>Skac reports a case of mania produced by tobacco, and Continental literature contains other observations.

The skin is usually muddy in color, and the mucous membrane of the tongue of an excessive smoker presents, according to some observers, the appearance as if it had been brushed over with nitrate of silver.

**Causes.**—Tobacco, when used to excess, does far more harm in some ways than others; and the purity of the substance and the method of its consumption greatly influence the troubles that may follow. <sup>4</sup>Anstie says: "There are a few whom no amount of care and skill exercised in taking the tobacco, nor any moderation in the dose used, can save from unmistakable poisoning whenever they indulge in it. These cases are rare, and they should be carefully separated from the evil results which are produced by mere unskillfulness in smoking." Chronic poisoning arises from certain bad habits, and these may be enumerated as: 1. Smoking when the stomach is empty. 2. Using several cigars in succession. 3. Inhaling the smoke of cigars or cigarettes. 4. Smoking only a pipe in which the nicotine has collected. 5. Swallowing the saliva. Among smokers it is found that the nervous effects are more easily produced in the early part of the day.

It is difficult to say just how much tobacco is harmful. In a case reported by <sup>5</sup>Gmelin, seventeen or eighteen pipes were smoked in quick succession by two men with fatal results.

The use of snuff by women in the manner known as "dipping," is happily becoming rare in this country. I have seen several examples of this kind leading to chronic poisoning. A stick, tooth-brush, or some such article, is dipped in fine snuff, and the gums and inside of the mouth are rubbed therewith. The toxic effects of tobacco are produced in a short space of time and are said to be pleasurable. I have found this custom to be prevalent among prostitutes, but it is by no means confined to them. In the case of a lady of refinement and social position, I found that a peculiar train of obstinate nervous symptoms were due to "snuff dipping," and search disclosed small parcels of snuff under her pillow and beneath the mattress of her bed.

Cigarette-smoking, which has increased to an incredible extent of late in this country, is much more apt to give rise to nervous symptoms, because of the tendency to almost constant indulgence, and the inhalation of the smoke.

<sup>1</sup> Manual of Psychological Medicine, p. 100.

<sup>2</sup> Annual Report of Philadelphia Hospital for the Insane, 1880.

<sup>3</sup> Ed. Med. Journal, Jan., 1856.

<sup>4</sup> Stimulants and Narcotics, p. 138.

<sup>5</sup> Reported by Woodman and Tidy, p. 379.

**Pathology.**—According to <sup>1</sup> Anstie, tobacco is a narcotic-stimulant, and he classes it with tea and coffee. The poisonous effects, as agreed by most authors, are excited in two ways: 1st. In interfering with the pulmonary circulation, retention of carbonic acid gas, and blood-poisoning. 2. A direct influence from the nervous tissue itself. The motor-nerves seem to suffer abasement of function, though the muscular irritability is not disturbed.

There seems to be some doubt as to the poisonous agent in tobacco. Vogel says that the toxic properties of tobacco-smoke are due to the presence of sulphide and cyanide of ammonia. <sup>2</sup> Eulenburg could not find a trace of nicotin (Woodman and Tidy), but he and Vohl believed the poisonous substance to be pyridin ( $C_5H_5N$ .) and parvolin ( $C_9H_{13}N$ .) <sup>3</sup> Huebel, however, has found the amount of nicotin in one cigar sufficient to produce convulsions and death in a frog.

There is undoubtedly in tobacco-smoke a certain amount of nicotin and other alkaloids in combination with alkaline bases. In gouty subjects, therefore, the use of tobacco cannot fail to be beneficial, when smoked in moderation.

In small quantities tobacco slightly exhilarates and increases the action of the heart, and one cigar may effect a prompt increase of thirty or forty pulse-beats—a secondary depression follows, however.

<sup>4</sup>Headland ascribes the comparatively light narcotic effect of tobacco to its prompt elimination by the kidneys, and says: "It is only not a poison because slowly taken into the system in small amounts and eliminated *pari passu*." In those individuals in whom, through disease of the excreting organs, the poisonous elements are not promptly removed, the production of nicotinism is much more prompt. The occurrence of vertigo is probably often due to a cumulative effect which occasions cardiac weakness. The cerebral effects of prolonged nicotinism are occasioned by the continued malnutrition of the brain tissue.

**Prognosis and Treatment.**—Nearly all the alarming symptoms can be immediately moderated or cut short by prompt discontinuance, and recourse to nux vomica or its alkaloid. The analysis of tobacco by <sup>5</sup>Schlössing and others, with regard to the quantity of nicotine has some bearing upon the evil effects attending its immediate use.

In 100 parts of Virginia tobacco Schlössing found 6.87 parts of nicotine. In the same quantity of Kentucky tobacco there were 6.09; in French tobacco, 4.94—7; Maryland, 2.29; Havanna, less than 2. In dry snuff there is 2 per cent.; in moist, 1.3.

Those who use tobacco are rarely inclined to acknowledge its bad effects but to attribute them to other causes; but, as Taylor says, "The argument that cases cannot be adduced to show direct injury to health proves too much—for a similar observation may be made of the habit of opium-eating."

<sup>1</sup> Stimulants and Narcotics, p. 100.

<sup>2</sup> Vierteljahrsschrift f. Ger. Med. N. F. xiv., p. 249, and Woodman & Tidy, p. 379.

<sup>3</sup> Centralblatt, Oct. 5, 1872. <sup>4</sup> Action of medicines, p. 269. <sup>5</sup> Quoted by Taylor, p. 771.

For the person who presents decided nervous symptoms traceable to tobacco, no better treatment can be suggested than the continuous use of a tonic containing iron, quinine, and strychnine,—such, perhaps, as the following:

R.—Strychniæ Sulphas,.....gr. i.  
 Quiniæ Sulphas..... ʒi.  
 Tr. Ferri. Chloridi..... ʒv.  
 Acidi Phosp. dil. }  
 Syr. Limonis..... } aa ʒij.

Sig.:—One teaspoonful in water thrice daily.

Strychnine alone, in small, repeated doses, or perhaps combined with digitalis, is useful. In amblyopia many authors, among them Webster, recommend the hypodermic use of strychnine. From 1-60 to 1-24 gr. may be given at a dose.

## HYDROPHOBIA.

**Synonyms.**—Rabies canina; Paraphobia; Lyssaphobia (?).

The name adopted to express that form of nervous trouble which sometimes follows the bite of a rabid animal is an evident misnomer, as the definition of the term signifies “a dread of water.” As this is but one symptom, and by no means a constant one, the first synonym is much more expressive and appropriate, and is in every way preferable to that in general use.

**Symptoms.**—1. *Period of Incubation.*—After the receipt of the bite, which may produce an extensive wound, or, as is the case sometimes, an insignificant scratch, a period of time extending from a few months to several years may elapse before the appearance of the second stage. The wound may heal by first intention, giving rise to no inconvenience, or there may be redness and neuralgic pain. A history of this kind is usually given by the patient, and is based upon an exaggerated statement of the actual facts, which arises from a disordered imagination, while his story of the accident and of his subsequent symptoms is tinged with a decided flavor of romance. Nervous derangement dependent upon fear, digestive disorders, mental worry, and others of the same category, generally characterize this first stage.

2. *Period of Invasion.*—At the end of the period of incubation, the first alarming symptoms noticed are those connected with the cicatrix, which becomes painful and tender, and at the same time there are pains which dart along the nerves in the vicinity. There are next generally headache and a sense of epigastric oppression, with constipation, broken sleep, and a feeling of general discomfort. At the end of two or three days, during which the patient suffers intensely, we may expect the appearance of the next stage.

3. *The Period of Development.*—With aggravation of the symptoms just enumerated, we find added thereto a sense of constriction about the throat, irregular and quickened respiration, rigidity of the muscles of the neck, discomfort in deglutition, and spasms, which begin in the muscles of



the throat and back of the neck, and gradually invade those of the back. The spasms give rise to much pain, which is sometimes spinal and at others muscular. The patient is at this stage delirious and flighty, and generally has delusions in which dogs play an important part. The difficulty of swallowing, which next follows, is not so great when solids are taken. Fluids, on the contrary, seem to produce an aggravation of the spasms, and the mere sound of splashing or trickling water will excite a convulsive seizure. To add to the sufferings of the patient, there is excessive thirst, which is very distressing. His face becomes dusky, and his eyes prominent and wild. He tosses from side to side if placed in bed, the saliva running from the angle of the mouth in a viscid stream. Towards the end of the disease this secretion becomes thicker and mixed with mucus, and it collects in the trachea and bronchi. These symptoms may last two or three days, while in the meantime the reflex excitability becomes so great as to precipitate a convulsion under the least stimulus. The pulse is rapid, the headache more severe, the air-passages become filled, and respiration is greatly interfered with. The convulsions are readily produced by blowing upon the patient, or by jarring him, or even by slamming the door. At this stage he becomes partially unconscious, is quite delirious, and very much agitated. Previous to death there is a marked rise in the temperature, and in one case I saw, the history of which I shall presently relate, the temperature rose to  $103^{\circ}$ , and I believe there was even a subsequent rise.

Death occurs in two or three days in most cases, but it may be delayed a day or two longer. Incontinence of urine and feces precedes the end; the immediate cause of death being asphyxia from spasmodic stenosis of the larynx, or obstruction of the air-passages by mucus. I had the privilege of seeing one case at the request of Dr. Augustus Viele, of this city, which was subsequently reported by Dr. Hadden.<sup>1</sup>

Through the courtesy of Dr. Hadden and Deputy Coroner Leo, I was also enabled to observe the *post-mortem* appearances of the brain and cord. Dr. Hadden describes the case so minutely that I shall mainly use his own words.

"On the 24th ultimo, at 8.30 P. M., I was called to attend a young man named Wm. McCormick, residing at No. 309 East 51st Street, a native of this city, aged 26 years, athletic in appearance, of usually good health, nervous temperament, and of moderately temperate habits; by occupation a driver of an express-wagon. He was in bed, complaining of nervousness, soreness in his neck and throat, strange feelings of tightness around his chest. His countenance was anxious, pupils of his eyes were dilated, and his general appearance was like one who was in fear of impending danger, and not in extreme pain. He told me that his throat was so sore that he could not swallow anything—not even water. This, he thought, was due to some simple medicine he had taken, and not to any serious ailment. I noticed his throat was not swollen on the outside, and that his

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<sup>1</sup> Journal of Psychological Medicine, May, 1870, p. 80.

voice was whining, and unlike a person suffering from any ordinary soreness within. I, however, examined his throat within, but found nothing to account for this difficulty; it was perfectly healthy in appearance. His pulse, respiration, and temperature were normal, excepting an occasional sigh. I observed, also, a little disposition to hack and spit, but in no way troublesome. He complained also of thirst, but said he could not drink, he knew, for the very sight of water made him shudder. I told him his throat was not sore, and urged him to try. He assented, and water was accordingly brought, which, at sight, caused a violent spasm. He threw himself around in the bed, forward and backward, and told the party to take it away at once, as it would kill him. He immediately afterwards called for the goblet, and said he was very thirsty and must drink, seized it, and with a violent effort succeeded in taking a single swallow, which was followed by a severe convulsive shudder and contraction of the muscles of the neck and chest." Dr. Hadden ascertained the fact that he had been bitten by a dog, and then inquired about the symptoms antecedent to his visit. "Wednesday and the two preceding days he was complaining of general lassitude and nervousness; had not been able to sleep at night; was thirsty, and had drunk a great deal of water; had eaten but little; appetite very poor, and on Wednesday afternoon he seemed to be growing worse. He went out upon the street, but soon returned, saying that it was very chilly, and he could not stand the air at all. While taking a cup of tea at 6 P. M. the same evening, he first showed signs of difficulty in swallowing. Shortly afterwards, as he was going to the kitchen, he was met by a draught of cold air, which so staggered him that he nearly fell; he then went to bed, where I found him. After giving the necessary caution to the family, I ordered fifteen grains of bromide of potassium to be given every hour. I left, and returned at 10.30 P. M. . . . Found him in about the same condition I had left him, only his pulse was irregular, and his spasms more frequent. The saliva was a little more troublesome, and he also could not swallow without great difficulty. I was called again at 2.30 A. M., the messenger stating that the patient had become very violent, and that they were unable to restrain him. I went immediately. . . . Found him in a frightful state of excitement; had broken down the bed, and was struggling with his attendants to get at liberty. He was shouting and crying out to them to let him go, and called for water, which, when brought, he could not drink. His mind was clear, and he knew all those around him; was spitting a viscid saliva, but was careful not to spit upon any one, not even on his clothes. It was so abundant that his attendants were obliged to wipe it from his lips. Dr. Leavitt and myself, after viewing the case in all its aspects, concluded to inject in the tissues of the leg half a grain of morphine and one-sixty-fourth of a grain of atropine in solution, which was done at 3 A. M. by Dr. Leavitt. We carefully watched the effect till 3.30 A. M., when, his violence having in no way abated, another injection was given in the same part of three-eighths of a grain of morphine and one-eighth of a grain of atropine, which in some degree produced the characteristic effect of morphine, and very clearly the appearances of the atropine; for, notwithstanding he was struggling violently, the saliva, which had been very troublesome, was completely dried up; so much so that the patient remarked that he was very thirsty, and his 'mouth felt as if he had been chewing a brick.' Fifteen drops of chloroform were then injected, with no effect whatever, unless to weaken his already weak and frequent pulse.

At 4.15 A. M. three-eighths of a grain of morphine were a gain introduced under the skin without atropine. This quieted the patient, so that he was easily restrained, and he remained in this condition from 4.30 till 10 A. M., when the effects had so far passed off that the attendants were alarmed at his violence and the abundance of saliva that he was spitting from his mouth. At 10.15 A. M. three-eighths of a grain of morphine in solution were injected in the tissue of the thigh, which served to temper down the increasing violence of the spasms, but did not stop the flow of saliva. I accordingly, at 10.45 A. M., injected three-eighths of a grain of morphine and one-fortieth of a grain of atropine, which had the desired effect of producing the quieting effect of the morphine and the specific effect of the atropia on the salivary glands. The poisonous effects of the morphine and atropia were at no time apparent. He died at 4.15 P. M. June 26, 1874, about twenty-four hours after the first spasm."

I saw him at three o'clock on the afternoon of the 26th day, and found him lying upon the floor bound with twisted sheets, the ends of which were held by his attendants. He was very violent, and, though there were no very marked convulsions, he seemed to be quite rigid, and his forearms were flexed during most of the time. He was semi-comatose, and groaned occasionally, but took no notice of those about him, and did not speak. His respirations were quick, and there was a rattling sound produced in his throat with each expiration and inspiration. A quantity of quite thick mucus and saliva was spat up during my visit, and there seemed to be a very free secretion of this substance. The pupils were widely dilated, and as far as I could judge there was no marked elevation of temperature.<sup>1</sup>

Recent cases of hydrophobia have been reported by Francois,<sup>2</sup> Edwards,<sup>3</sup> Smith,<sup>4</sup> and Hanscom.<sup>5</sup> The case of the latter is so interesting and so graphically detailed, that I shall take the liberty of giving it in full.

On the morning of the 20th November a good-natured pet spaniel, which had never been known to snap at any one, suddenly and without any provocation sprang at his mistress. His master whipped him, and he was left in the cellar of the house until the time for his dinner. When eating it in the company of a pet cat, as he had been accustomed to, without ever having molested her, he suddenly seized the cat and threw her across the room. The owner reached out his hand to catch the dog, when the latter caught him tightly by the wrist and inflicted a deep wound, biting him three times; the skin became lacerated while making an effort to shake him off. It was supposed at the time that the dog was

<sup>1</sup> In this case the newspapers were filled with sensational accounts of the patient's illness, and an attempt was made to prove that the dog was not mad. It is needless to say that such was probably not the case, and it is to be regretted that the dog was never found.

<sup>2</sup> Bost. Med. and Surg. Journal, May 17, 1877.

<sup>3</sup> Ibid., March 15, 1877.

<sup>4</sup> Ibid.

<sup>5</sup> Ibid., April 19, 1877.



irritable from the whipping which he had received in the morning, and, as he expected another for snapping at the cat, defended himself by biting. Half an hour after, the patient applied to me for treatment, and believing it to be too late for incision or cauterization to be effective, and as there was no history of hydrophobia, I dressed the wound with a solution of carbolic acid. It healed readily, and the patient attended to his business as usual in four or five days. Soon after the infliction of the bite the dog disappeared and he did not return for thirty-six hours; nothing could be ascertained of his whereabouts or of his behavior during that time. When he returned he was very much exhausted, and had the appearance of having been severely beaten. From what I can learn of those who saw him he gradually grew weaker, apparently losing the use of his legs, especially the hind ones, which he would drag after him. He died quietly, with his head in the lap of his mistress, without having had a convulsion, excessive flow of saliva, or tremors. On the 13th day of January (fifty-four days after the injury), the patient began to have shooting pains in the forearm, but not especially localized. They did not radiate from the cicatrix, and there was no change in the appearance of the latter. On the following day the pain had increased so much that he required one-sixth of a grain of morphia to relieve him; it was given subcutaneously, and was repeated the next morning. After that there was very little pain in the arm, and no appreciable change in the pulse or temperature. He was despondent, and stated on the morning of the 15th that 'he felt sick and used up all over;' he was obliged to go to bed in the afternoon, and then for the first time began to have some difficulty in swallowing. This symptom was not manifested by an attempt to drink water, but during an effort to swallow some herb tea which he was accustomed to take when ill, and which he believed would relieve his bad feelings. There was no trismus; he was quiet and inclined to doze. At 5 P. M. Dr. H. H. A. Beach saw the patient with me, and agreed that the history of the case in connection with the symptoms then existing indicated the probable development of hydrophobia, and an unfavorable prognosis was given to the patient's brother, who promised not to communicate it to the patient or his friends until the disease should be fully declared. His pulse at this time was 102, and the temperature in the axilla 102° F., face flushed, tongue coated. The cicatrix presented no unusual appearance, nor was it tender. A dark room was agreeable to him, but on raising the curtains the light did not disturb him in the least. He was perfectly rational, and had some thirst, but no sore throat. He made an attempt to swallow a teaspoonful of milk, but was obliged to give it up from the moment that the fluid touched his lips. Immediately after this attempt unmistakable spasmodic contraction of muscles between the chin and sternum was observed. Mentally the patient was perfectly clear, and not disturbed by the unsuccessful attempt at swallowing fluids, but said he would try it again when he should be more thirsty. This symptom, excepting when he swallowed teaspoonful-doses of medicine, continued until his death. He was obliged to relieve his thirst by sucking ice and snow through a napkin. The air from a fan or from adjusting the bed-clothing caused a shudder. Occasional sighing was noticed after the second day; it grew deeper and more frequent until the end. When disturbed from any cause, his respiration was of a spasmodic character, so much so at times as to interfere with his speech.

On the following morning (the 16th) his pulse was 96, and mild de-

lirium first developed; this also continued until his death. He was easily controlled throughout the disease. He became very suspicious of the people about him, believing that they were attempting to make him the victim of practical jokes, then of being poisoned. One hallucination was continuous from the time that the delirium first developed: he thought that some one had thrown a dirty powder on him, and he was continually making efforts to shake it off from himself and his clothing. He was also very cross and dictatorial, but showed no disposition to snap or bite.

Between four and five P. M. on the 18th he began to have spasmodic contraction of the muscles of the chest, larynx, and throat; some of them lasted nearly a minute, and prevented him from taking an inspiration. He also had a profuse discharge of saliva sufficient to wet his clothing through from his chin down to his hips. The spasmodic contractions concerned in respiration exhausted him rapidly, and he died quietly at 8 15, while sitting up in a chair. This position became necessary from the fact that he could not lie on his side, and if on his back the saliva accumulated so rapidly that it obstructed his respiration. For the last twenty minutes before his death there was no spasm. He lived five days after the first general symptom. At no time was he disturbed by the sound of ringing bells or running water. Morphia in one-fourth-grain doses, and chloral and bromide of potassium in fifteen-grain doses of each at the same time were given as needed. Anæsthetics were not required. At the solicitation of his friends he was allowed to take a pill, the prescription for which was said to be one hundred years old and to have cost originally five hundred pounds. It had the reputation of curing and preventing many cases of the disease. No change in his symptoms could be attributed to its action, nor could its composition be ascertained. It was given as a placebo, on the chances that an hysterical element existed in this case; that whatever offered encouragement to the patient without the possibility of injury in his hopeless condition was justifiable, but so far as the evidence furnished by one case is of value its inefficacy was demonstrated. The permission of his friends for an autopsy could not be obtained. The particular symptoms of the disease which were not observed in the dog when seen might have existed during the thirty-six hours that he was absent.

The proximity of the wound to the ulnar nerve and its character (punctured and lacerated) suggested the consideration of tetanus as an explanation of the symptoms; the latter seemed to be fairly excluded, however, on the ground that delirium was continuous from the third day of the attack, and that at no time did trismus or any other form of tonic spasm exist; the profuse discharge of saliva was also corroborative of this view. The unquestionable existence of repeated attacks of laryngeal spasm; the fact that the symptoms developed after a considerable interval had elapsed from the date of the injury; that for three hours previous to his death, and after he became wholly unconscious, marked spasms of the chest and throat occurred at intervals of from three to five minutes; that death occurred as a result and within five days following the development of symptoms characteristic of the disease, reasonably offset a theory that the hydrophobic symptoms were simulated by an hysterical man.

In Smith's case the period of incubation was about two months, and the paroxysms were ushered in by vomiting, fear of water, and febrile symptoms. On the third day of the disease he became delirious, and on the

fourth died. The sound made by the patient, which is so often compared to the bark of a dog, was likened by the author to that made by a croupy child. In Edwards's case, the period of incubation was about five months. The injury was insignificant, but with the invasion of the disease there was pain in the cicatrix which extended up the arm. In this patient there was also dread of fluids, especially water. On the second day the convulsions began. The same day she spat up bloody mucus. At the end of sixty hours from the first local pain she died.

**Causes.**—The circumstances which concern the etiology are still enshrouded in mystery. Some authors are of the opinion that rabies may be communicated by a dog that is not mad, and cases are brought forward to prove this theory. I cannot agree with this, for it seems to me highly improbable that there should be so few cases of this disease if the bite of a non-rabid animal can inoculate an individual. Bouley states that in no way can the disease be transmitted other than by inoculation with the saliva. In this statement he receives the endorsement of Magendie and others. Another point remains to be answered, and this is in regard to the transmission of virus from one person to another without the second person being bitten. Fleming has given an example which shows that this may take place.

In the spring of the present year I was subpoenaed to serve as a jurymen in the case of a boy who had died of rabies. At about the same time another death occurred which the attending physician said was simply the result of fear, and not of hydrophobia. A careful inquiry and examination of witnesses revealed the following history, which I think proved beyond a doubt that the cause of death in both cases was the bite of a rabid cat. This cat had found her way into a stable on Thirty-fourth Street, and had bitten a horse. This horse afterwards died in convulsions, and from all I could learn the cause of death was hydrophobia. In an adjoining yard the cat bit one of the boys, who also died, and in a few days afterwards bit the other boy, whose inquest we attended. Both of these victims died within a short time of each other. In one of these cases there was but a slight scratch.

**Morbid Anatomy and Pathology.**—Clifford Albutt,<sup>1</sup> Meynert, Elder,<sup>2</sup> and others have made autopsies, and still there seems to be very little light thrown upon the pathogeny of the disease. Albutt found enlargement of vessels in the cerebral convolutions, pons, medulla, and spinal cord, and granular disintegration. Elder found absolutely nothing; and the results of the search of Lockhart Clarke who examined parts of the brain, medulla, and cord, were equally negative.

Kolesnikoff<sup>3</sup> reported the appearance of the nervous centre in ten dogs that had died of hydrophobia. "The parts examined included the hemispheres, corpora striata, thalami optici, cornua ammonis, cerebellum, medulla oblongata, spinal cord, the sympathetic and vertebral ganglia. The

<sup>1</sup> Med. Record, i. 22.

<sup>2</sup> British Med. Jour. vol. ii. 1874.

Centralblatt für Med. Wissen., No. 50, 1875. Abst. Phil. Med. Times, Feb. 5, 1876.



most marked changes were observed in the two latter, and were as follows: 1. The vessels were enlarged, choked with red blood-corpuscles; occasionally, extravasated red corpuscles and round indifferent elements (probably white corpuscles) were found in the perivascular spaces. The walls of the vessels were here and there filled with hyaloid masses of various forms, which occasionally extended into the lumen of the vessels, and closed this as a thrombosis would. Not far from these masses collections of white and red blood-corpuscles could be observed, the latter deprived of color. They could be seen also in all stages of metamorphosis into hyaloid globules. 2. In the pericellular spaces of the nerve-cells could be observed collections of round indifferent elements, whose penetration, to the number of five to eight or even more, pressed out the protoplasm of the cells. This penetration of the elements spoken of was frequently sufficient to change the form of the nerve-cells, giving them at different times a sac-formed, bulged, or flattened-out appearance. Further, the nucleus was sometimes pushed towards the periphery of the cell and surrounded by many round elements. In other cases, only groups of round (indifferent) bodies could be observed in place of the nerve-cells. In isolated nerve-cells the changes described could also be observed."

The body of Dr. Hadden's patient was examined by the deputy coroner and several physicians, among whom were Drs. Clymer, Hammond, Cross and myself. The calvarium was removed, and great congestion of the meninges and brain was observed. The sinuses were much engorged, but there was very little effusion either upon the surface of the brain or in the ventricles. The lower surface of the brain appeared to be slightly softened in patches, but there was nothing else to attract attention, except it might perhaps have been a great hardness of the pituitary body. The internal viscera were all hyperæmic, but there was no other morbid appearances. The larynx and trachea were found to be very much injected, and the latter contained a quantity of frothy mucus. Dr. Willis has found the blood of persons who have died from this disease to be very fluid and of a dark color. Dr. Shattuck and Fitz<sup>1</sup> have published the notes of an interesting case of hydrophobia treated unsuccessfully by them. An immense amount of curare was given, about four grains within six hours, without any of the physiological effects being produced, though the drug was of good quality. Dr. Fitz's subsequent examination is of so much interest and so full that I present such parts of it as relate to the change in the nervous tissues:

"While exposing the spine the surrounding tissue seemed to contain less fluid than usual. No abnormal appearances were observed in the membranes of the spinal cord, or upon the surface of sections made across the latter at intervals of an inch throughout its length.

The calvaria was readily separated from the dura mater, both the bone and the membrane presenting no unusual appearances. The longitudinal sinus contained a soft gelatinous clot, only partially filling the

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<sup>1</sup> Boston Medical and Surgical Journal, Aug. 28, 1878.

cavity. The pia mater was occasionally spotted and streaked from fibrous thickening, and was unusually injected over the greater part of the convexity of the brain, the vessels being often varicose. The meshes contained a considerable excess of clear fluid, and the membrane was readily detached from the brain. On section of the brain no unusual appearances were observed in the ventricles or cerebral substance beyond abundant puncta cruenta.

The chief interest naturally centered in the possible condition of the nervous system, and the spinal cord, medulla oblongata, and portions of the cerebral convolutions were preserved in Müller's fluid for the purpose of microscopical examination. Positive results were obtained from the medulla alone; it should be stated, however, that the cord was perfectly hardened, so that the sections obtained from it were comparatively useless. The changes found in the medulla were observed throughout its length, and were most commonly met with in the posterior portion, especially in the immediate vicinity of the floor of the fourth ventricle. The alterations were most extreme in that part corresponding with the *calamus scriptorius*. The appearance most frequently met with was infiltration of the adventitia of the veins with small, round cells, both large and small veins being affected. So abundant was their distribution that upon longitudinal section the wall of the vessel seemed to be paved, as it were, with these cells. As a rule, the vessels thus modified were distended with blood, and it seemed probable that the observed changes were pathological, as the vessels in other parts of the medulla did not present such an appearance. The injection of the veins was so complete at times that their section was of a dark-brown color and quite opaque, the individual corpuscles being indistinct, and the condition deserved to be spoken of as a thrombosis. It was evident from transverse sections that the different cells were not simply adherent to the inner surface of the vessel, but were actually within the wall, nor was there any evidence of an increase in the relative proportion of white to red blood-corpuscles.

Another appearance often met with was that of hæmorrhage. In general the extravasated blood was found within the perivascular, particularly venous, spaces. The sharply-defined outline of the corpuscles and the absence of granules of blood-pigment indicated that the hæmorrhages were recent. Transverse sections of the injected vessel, with its wall infiltrated with round cells, and a perivascular accumulation of red blood-corpuscles, were often met with. In none of the sections were ruptures of the vessel wall seen. At times the wall was somewhat collapsed, the contents correspondingly less, while around the vessel a considerable hæmorrhage was apparent. The hæmorrhages were usually limited to the perivascular space, the blood rarely having made its way between the nerve fibres or into the gray matter.

Finally, an appearance was sometimes met with which may be spoken of as a miliary abscess. Occasional minute agglomerations of indifferent cells were seen, but their relation was such as to suggest their prob-

able origin from sections through limited portions of the infiltrated adventitia already referred to. In two instances, however, actual abscesses were found,—one within a convolution of the olivary nucleus, another in the immediate vicinity of a pigmented ganglion cell in the upper part of the medulla. The former was a larger, and it was found in a part where none of the cellular infiltration of the vessels already mentioned was observed.

In brief, then, the alterations were a diffuse cellular infiltration of the adventitia of the veins, venous injection and thrombosis, perivenous hæmorrhages, and miliary abscesses."

The question to be answered after all is, whether this affection is a primary disorder of the nervous centres or whether it is the result of general blood-poisoning. I am inclined to accept the latter theory, as the array of facts is too meagre to permit any positive assertion as to its nervous origin. Like other disorders, not essentially nervous, there is a period of inoculation, or incubation, of invasion, and development. I think, then, that in this respect this disease, as well as tetanus, resembles closely some of the exanthemata.

**Diagnosis.**—It is important to bear in mind the fact that a great many so-called cases of hydrophobia are not this disease at all, and that certain forms of hysteria bear to it a close resemblance. Fright may act so powerfully upon the nervous system that a train of symptoms may be produced very much like those of the genuine affection. A case of this kind occurred at Bellevue Hospital a year or two ago, in which the symptoms counterfeited those of the real disease in every respect, and the patient finally died. It was found that the individual had not only never been bitten, but that he actually died of fear, his imagination having been stimulated by the sensational articles in the newspapers. Dr. J. W. S. Arnold, of the University, who examined the brain and cord, was unable to find the slightest indication of any morbid change. The only other conditions from which we may be required to make a differential diagnosis are tetanus, Calabar bean, and picrotoxin poisoning. In the former there are many points of resemblance, and occasionally a dread of liquids and a difficulty in swallowing. In tetanus, however, the *risus sardonicus* is present, the spasms are tonic, and there is opisthotonos, and the mind is clear to the last.

In poisoning by both agents, to which I have alluded, the rapidity of their action is conspicuous, and a dose of either would carry the patient off in a few hours, more or less. In picrotoxin and Calabar bean poisoning, there are many of the symptoms of hydrophobia, such as clonic spasms, frothing, rise of temperature; but no dread of water, nor delirium.

Epilepsy may resemble hydrophobia, but it is only when the attacks are numerous and closely connected that such a mistake could possibly occur.

Marbaix<sup>1</sup> "gives a case of epileptiform convulsions more or less resembling hydrophobia, in a man who had been bitten four days before by a

<sup>1</sup> Presse Méd. Belge, 1869, 237.



cat; they were accompanied by delirium and hyperæsthesia of the optic nerve, a stray light thrown across his eyes causing a convulsive attack. The shortness of the incubation, the blueness of the face, without the 'vultueuse' expression characteristic of hydrophobia, the delirium, and the melancholy, not exalted, condition, combined with a history of an epileptic attack a year before, prevented the case being looked upon as one of true hydrophobia."

**Prognosis.**—In true hydrophobia it is very bad. I believe there never have been more than one or two genuine cures reported; and if others have been claimed, it is probable that no rabies existed, but that the affection described was simply hysterical. The chance of inoculation seems to be a matter of interest, for of the reported cases in which individuals have been bitten, it has been found that about two-thirds of them subsequently developed symptoms of rabies.

**Treatment.**—We rarely see these patients until actual evidences of madness have appeared. If, however, we are fortunate enough to be called to the individual immediately after he has been bitten, we may either incise or cauterize the wound. It is well to ligate the limb as soon as possible, and then remove *en masse* the piece of the muscle which has been penetrated by the teeth of the rabid animal. Various writers recommend the cupping-glass, which should be applied to the excised part till it abstracts several ounces of blood from the wound. A pencil of nitrate of silver may be thrust into the punctures made by the teeth of the dog until they are well cauterized, and a strong solution (3ij-℥j) should be applied afterwards by means of a piece of folded linen, which is to be covered by oil silk.

I am convinced that no remedy can do good where the disease has already appeared, except, perhaps, curare, which has been tried; and in one case, where it was prescribed by Dr. Austin Flint, Sr., it is said to have saved the patient's life.

The case must be desperate, however, when this powerful substance is resorted to, for its preparation is not always the same, and no two specimens are of the same strength. It has been injected hypodermically in doses of one grain.

Offenberg<sup>1</sup> reports the cure of a girl of eighteen. She received at first hypodermic injections of morphine and chloroform, but there was no improvement in her condition. Seven hypodermic injections, aggregating three grains of curare, were afterwards given in the course of six hours. The muscular disturbance subsided at once, and there was ultimate recovery. The convulsions were succeeded by paralysis, which gradually disappeared.

Hot baths have been recommended, but I cannot find that they have ever cured a case of this kind.

## HYSTERIA.

**Synonyms.**—Hysterie (Fr.) Muttersucht (Ger.) Vapors.

<sup>1</sup> Wien. Med. Presse, 1876, No. 1.

**Definition.**—It would be almost impossible to give a concise definition of this most protean of nervous affections, for it simulates a multitude of organic and functional diseases so perfectly, that the task of considering it in any systematic manner would be attended with great difficulty. The nervous system in this respect is like the “general utility” actor. It plays the most varied parts. Sometimes we are presented with a hemiplegia or paraplegia, and at others with contractures which seem to be the result of organic disease, so permanent and intractable do they appear. Convulsions, anæsthesia, urinary and other troubles of a more or less grave character, swell the list, until we are almost inclined to look upon it as a “disease of the Devil,” and cease to wonder at the credulity and superstition of those who believe in demoniac possession and witchcraft. Confining ourselves as closely to the subject as possible, we conclude that hysteria is a disease of an emotional character chiefly among women, in which the symptoms are rarely the same in any two instances, but among a large number of cases there can be noticed a certain similarity.

**Symptoms.**—These symptoms may be grouped as *sensorial*, *motorial*, and *visceral*. *Sensorial* symptoms are of three kinds; hyperæsthetic, anæsthetic, and mental. Hyperæsthesia, though much more common than anæsthesia, is not so marked. Large areas of hyperæsthesia may be detected by careful examination, though the patient usually saves this trouble, for she calls attention to the weight of her clothes, the pressure of some fold of her underwear, or the contact of some very light substance which is pronounced unbearable. The external organs of generation are extremely sensitive, and the slightest touch of the finger or speculum produces a spasm and great agony. Coition is impossible, and one patient called my attention to a horrible shooting pain which occurred whenever her husband approached her. Hyperæsthesia about the nipples, at the end of the coccyx, and in other parts of the body, is alluded to by various writers. Charcot has directed attention to the prominence of these; and Briquet has described fixed pains of the abdomen which he called *cœlalgie*, and of 450 cases he found 200 presenting this symptom. They were hypogastric and iliac, but more commonly the latter. These have sometimes been mistaken for the pain of peritonitis; there is, however, no tenderness, but simply superficial elevation of sensibility. The patient often calls attention to vague pains in different parts of the body, of a transitory, and sometimes permanent character. She complains of strong light and loud noises, and insists upon perfect quiet, although she will herself talk and cry in a very noisy manner. All of her pains are increased when her attention is concentrated upon them, but when her mind is diverted she will bear very rough treatment without complaint.

Neuralgic pain, a familiar variety being the *clavus hystericus*, is a common form of complaint. Various local pains are also experienced, and these, among others, include alterations in sensibility which simulate lumbago; indeed, a very constant hysterical complaint is backache, which the patient generally attributes to the kidneys. A most interesting form

of hysterical dysæsthesia has received mention from Skey, Paget, and others, and is very often mistaken for rheumatism. The joints are neither swollen nor red, however. M. Meyer,<sup>1</sup> in an interesting article upon the subject, gives the leading points in diagnosis as follows: "1. The neuralgia is of a diurnal form entirely. 2. Light pressure of joints produces pain, but comparatively violent handling is not at all painful. 3. The temperature of the affected joint undergoes variations. 4. There is no loss of substance of the muscles of an unsound limb. 5. The cure is usually spontaneous." The mental disturbances are of the most interesting character, whether expressed by transient emotional excitement or apparent prolonged unconsciousness. Examples of the lighter grades are too familiar to need description, and it is only necessary to allude to the outbursts of immoderate laughter or crying which occur when there is no reason for either emotional elation or depression. Such individuals may indulge in laughter at church or at a funeral, and, while perfectly aware of the impropriety of their conduct, will be utterly unable to restrain themselves. Illusions, hallucinations, and even delusions are evidences of a very irritable condition of the nervous centres, as are ecstasy and mental excitement of various kinds, such as belief in impending calamity or death. The involuntary use of foul words and gestures, and a remarkable eccentricity of behavior, are additional suggestions of a disordered state of the emotions. Wynter,<sup>2</sup> in his excellent little book, thus alludes to a condition which, after all, is but a manifestation of hysteria.

"There is a terrible stage of consciousness in which, unknown to any other human being, an individual keeps up as it were a terrible hand-to-hand conflict with herself when she is prompted by an inward voice to use disgusting words, which, in her sane moments, she loathes and abhors. These voices will sometimes suggest ideas which are diametrically opposed to the sober dictates of her conscience. In such conditions of mind, prayers are turned into curses, and the chastest into the most libidinous thoughts."<sup>3</sup> The will is quite weak, while the emotions, far from being held in abeyance to the extent which they are in health, respond to trivial ideational impressions. The hysterical person firmly believes herself to be the subject of various disorders of a greater or less serious character; is hopeless; believes in a speedy fatal termination of her imaginary trouble; and can only be convinced of her mistake by fear of the remedy suggested, or by some strong appeal to her appetite or comfort. While in a state which may sometimes appal the observer, the patient declares her inability to walk. If, however, some powerful excitement be produced, such as an alarm of fire, she quickly recovers the use of her legs. I have recently seen a most interesting case of hysterical torticollis, in

<sup>1</sup> Berliner Klin. Woch., 1874, No. 26.

<sup>2</sup> Borderland of Insanity, p. 3.

<sup>3</sup> Hysterical girls and women occasionally evince a depraved appetite, eating all sorts of extraordinary things. The school-girl habit of eating slate-pencils is an example of this. I have personally observed this evidence of hysteria on many occasions. A young lady recently under treatment ate enormous quantities of nutmegs. The morbid appetite of pregnancy is probably an hysterical disorder.



which the patient refused to turn or raise her head. I quietly seated myself at her other side, and engaged her attention so fully that after a while she turned her head and talked for some time; and it was only when I referred to the subject of her troubles that she quickly resumed her original position, and I could not persuade her to change it. She may at times believe that she is deaf or dumb, and remain in such an uncomfortable condition for years, punishing not only herself, but making all about her uncomfortable.

One of the most striking mental characteristics of the hysterical woman is her utter want of confidence in herself. She relies upon all those about her, and goes to her physician at all hours and with no object in view except the need for sympathy. She often has an impending dread of some calamity, and requires constant reassurance. If the physician could give her the belief that she could control her own emotions and conquer, much might be done. She even may know how unsubstantial are her symptoms—her paralysis, for instance, but she says “I cannot help it; I have every desire to move my leg, or my arm, but I know that I cannot.”

Hysterical anæsthesia has received a great deal of attention of late years from the French observers, especially from Charcot, as well as Piorry and Gendrin. Briquet<sup>1</sup> has found that this condition occurs more frequently on the left than upon the right side. It may be superficial or deep, even affecting the muscles and bones. Reynolds has found it limited often to the back of the hand or foot, or about the mouth and nose. The vaginal canal and the lining mucous membrane of the mouth are also places where there may be loss of sensation. Hysterical hemianæsthesia does not differ from that due to cerebral hemorrhage so far as the symptomatology is concerned. The same regions are affected and the same complicated amblyopia takes place. Taste and smell are unilaterally involved. Hysterical anæsthesia not rarely follows, or comes on during a convulsive attack, and lasts for a variable time. It may subside in a few hours, or continue for months at a time. During its existence the most violent stimuli will fail to restore sensibility; and I have often used powerful counter-irritants, electricity, or even the hot iron, without any response whatever. The loss of sensation may extend more deeply, so that the underlying muscles may be utterly without sensation. This peculiarity probably explains the insusceptibility to pain spoken of by Carré de Montegeron. The Jansenists or Convulsionnaires “became so wrought up by religious excitement that they fell, twenty or more at a time, into violent convulsions, and demanded to be beaten with huge iron-shod clubs, in order to be relieved of an unbearable pressure upon the abdomen. One of the brothers Marion felt nothing of the thrusts made by a sharp-pointed knife against his abdomen.”

Not only may there be analgesia, but loss of appreciation of heat or cold, and the surface may become blanched and white, and the skin even bloodless. Brown-Séquard has demonstrated the absence of blood; a fact

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<sup>1</sup> *Traité Clinique et Thérapeutique de l'Hystérie*, Paris, 1859.

which has an historical interest in connection with the tests of the early religious enthusiasts. Charcot alludes to the epidemic of St. Médard, when the cut of a sword failed to produce any flow of blood. The temperature of the anæsthetic spot is sometimes lowered two or three degrees, and varies in different regions. There may be anæsthesia of the mucous membranes of the mouth, the pharynx, and larynx; or the organs of special sense may be implicated, and a resulting amaurosis, amblyopia, or deafness ensue. In a paper upon "Hysterical Affections of the Eye," by Dr. Geo. C. Harlan,<sup>1</sup> of Philadelphia, attention is directed to retinal anæsthesia and various hysterical disorders of an interesting character.

"Almost any derangement of vision may be counterfeited. A little girl of eight years complained that every object that she looked at seemed covered with diagonal white lines, the direction of which she indicated with her finger. As the ophthalmoscope revealed a normal fundus, a favorable prognosis was given. This was made more positive the next day, when the white lines changed to blue, and was justified by the early disappearance of the difficulty.

"In the second class of cases we have more or less retinal anæsthesia, with anomalous and variable symptoms, changing, perhaps, at each examination.

"In the third class of cases the parts affected have been the retina, the muscle of accommodation, the external muscles of the eyeball, and the elevator of the upper eyelid.

"It is not very uncommon to meet with patients who have apparently perfect eyes and full acuity of vision, but who say that the test letters become blurred and unrecognizable after they have looked at them for a few seconds. That this is due to an exhaustion of the sensibility of the retina which disables it from the sustained performance of its function, and not to an irregular action of the accommodation, is shown by the fact that it persists when the eye is fully under the effects of atropia.

As to color blindness in hysterical women, I think its importance has been exaggerated, and I have very rarely met with even the slightest affection of the color-sense, unless the hysteria has existed in connection with cerebral disease and hemi-anæsthesia.

Taste and smell are sometimes impaired, so that there is a greater or less extensive loss or a perversion, the patient declaring that natural odors are reversed, or that articles of food are tasteless.

The *Motorial* symptoms are numerous, and may be either of a sthenic or asthenic character. The more simple include spasms, violent gesticulations, and contractures: the more obstinate, paralysis of either a hemiplegic, or paraplegic, or even a local form, and chorea and convulsions, as well as various kinds of muscular incoordination. The individual may assume the most painful positions, the limbs being rigidly flexed or extended, and the face distorted by grimaces of the most absurd description. Sometimes there is torticollis, or spasm of some small group of muscles, or the muscular

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<sup>1</sup> Phil. Med. and Surg. Rep., August 12, 1876.

rigidity may even amount to opisthotonos, pleurothotonos, or emprosthotonos, and these forms of trouble are much more marked in conditions of hystero-epilepsy and hystero-catalepsy. The dependence of these motorial phenomena upon reflex excitement is their marked feature, slight peripheral irritations, uterine trouble, or sexual excitement of any kind, often being the origin of the affection.

The pharynx, larynx, and not rarely the stomach are implicated, so that difficulty of swallowing, loss of speech, and vomiting are resulting phenomena. Hysterical attacks of a convulsive character are met with sometimes, when the patient is apparently unconscious, but is in reality not at all so. There is slow respiration, which is scarcely perceptible, and small weak pulse. The legs and arms may be wildly thrown about, or rigidly extended, and there may be opisthotonos, while the skin is livid, and may be bathed in perspiration. A lighter grade of attack is frequently seen, in which the patient, after a period of excitement, screams, and falls to the floor (being very careful not to hurt herself); her muscles become contracted; she breathes heavily, froths at the mouth, talks incoherently, and berates those about her. She may cry, and in doing so sobs violently, sometimes catching her breath in an alarming manner, frightening her attendants and attracting sympathy. If left to herself and not noticed, she may fall asleep or gradually recover. The patient looks about the room during the attack, and is undoubtedly conscious of what transpires. One significant mark of hysteria, previously alluded to, is that, however much the patient throws herself about, she is always careful not to do herself injury. Pomme<sup>1</sup> was among the first to describe hysterical contractures, and later Gorget related a case of hysterical flexion of the thigh upon the pelvis which was supposed to be due to coxalgia. In hemiplegic contractures the upper limb may be drawn in to the trunk, the forearm is flexed at a right angle, the thumb is bent so that the point is buried in the palm of the hand, and it is covered by the other fingers.

According to Strauss,<sup>2</sup> extension of the upper limbs is quite rare. The lower limb is extended, so that the foot presents the appearance of talipes equinus, the toes having a claw-like appearance. The thigh is extended on the pelvis, and the whole limb is adducted.

Hysterical contractures of a permanent character may affect the body, either laterally or below the waist, or but one member may be involved. Charcot<sup>3</sup> relates a case in which the left leg was firmly extended. The foot presented the deformity of talipes varus, and the limb was very rigid, so that, by lifting it, the body could be moved without bending the knee. The contracture could be overcome by chloroform, but returned when its effects had disappeared. In this case the limb was agitated by a tremor, or "tremulation convulsive," as this author calls the movement. These hysterical contractures often last for years, and are cured spontaneously. Skey<sup>4</sup> relates a case which is quite interesting.

<sup>1</sup> *Traité des Affections Vaporeuses.*

<sup>2</sup> *Des Contractures*, Paris, 1875.

<sup>3</sup> *Op. cit.*, p. 307.

<sup>4</sup> *Hysteria, etc.*, London, 1866.



"In the year 1864 a young lady of 16 years of age was placed under my care under the following circumstances: For eight months prior to her visit to me, she had been suffering from inversion of the left foot, which was so twisted as to bring the point of the foot to the opposite ankle; in fact, at nearly a right angle with the foot of the opposite side. Her family consulted a surgeon of much experience in the treatment of distortion, and of orthopædic notoriety. The case was considered as an example of an ordinary distortion, and the foot was placed in a very elaborately made foot-splint, by the force of which it was made to approach a parallel relation to the opposite side; but it was an approach only, for no mechanism could retain it in a perfect position, the toes yet to some degree pointing inwards. A month elapsed, and the disease continued unchanged. A second orthopædic authority was then consulted in conjunction with the first, and as no new light was thrown on the disease by the combined opinions of the two, the same principle of treatment was recommended to be continued, and the mechanism was yet somewhat more elaborated, and thus the eighth month of the young lady's life passed away, during which no constitutional treatment was resorted to, and loss of exercise, for she walked, it was almost unnecessary to say, with great difficulty."

Skey examined the foot, and arrived at the conclusion that the inversion was too great to be due to the muscles alone, and discovered that those of the whole limb were involved; that the disease had appeared suddenly in a girl of 15 years, who was otherwise well and strong, and in whom there was no indication of acute local disease.

The apparatus was removed; a hearty diet, with tonics, was ordered; she was told to walk; and at the end of six months was invited to a ball, her foot being still deformed. She accepted an invitation to dance, and remained standing throughout the entire evening. She had been suddenly cured.

Hemiplegia and paraplegia of an hysterical character are sometimes met with, as well as local paralysis, but the face is rarely affected in hysterical hemiparesis, and the tongue never so.

The walk is quite different from that of organic hemiplegia, and the foot is simply dragged along and not swung, and there is an absence of that helplessness which is so characteristic of the serious trouble. Electric sensibility and contractility are not usually affected, though the former may be occasionally impaired. The cure is spontaneous, and there is never atrophy or any of the peculiar tissue changes of neuritis which generally follow hemiplegia from cerebral diseases. Paraplegia of the hysterical variety is rarely attended by any urinary or rectal trouble, and never by incontinence, and the muscles are well nourished and respond to electric stimulation. Some voluntary motion is possible in the recumbent position, and it is only when the patient walks that she shows her loss of power. Reynolds states that a peculiarity of the disease, which is familiar to all, is the fact that no amount of help can keep the patient from staggering or falling; she may be supported by strong arms, but she sinks to the ground, not, however, falling entirely, but regaining her position by a voluntary effort.

The patellar tendon-reflex is usually increased upon the paralyzed

side in hysterical hemiplegia. I have never found it to be diminished, but care should be taken to define the line between the paralysis, due to myelitis, with hysterical symptoms, and the hysteria, in which there is paralysis. I have referred to the former cases in a previous article.

The *visceral* troubles are a host in themselves. Not only may the patient complain of unbearable pains situated in the liver, stomach, and other organs, but there may be urinary affections of considerable importance. Two varieties of hysterical urinary derangement are spoken of by Charcot, one being ischuria, and the other a complete suppression, which he has called *oligurie*. In both cases the urinary passages are perfectly normal; in the first there is simple retention of urine in the bladder; and for a long time (amounting even to months or years) it will be found necessary to use a catheter.

Laycock<sup>1</sup> has called attention to this state of affairs, which lasts sometimes twenty-four or thirty-six hours, during the menstrual epoch. Charcot has found the condition to last even longer—sometimes for several days. This suppression of urine is occasionally accompanied by vomiting, and the presence of urea has actually been discovered in the vomited substances. This has been explained by the experiment of Brown-Séquard, who found that after certain forms of mutilation carbonate of ammonia or free urea was found in the intestines of animals, which settled the fact that there was a “supplementary elimination.” This same condition of affairs is not unusual in renal disease, and the odor of the breath and sweat is decidedly uriniferous. Vomiting of fecal matter is a rare symptom. There is in the majority of cases a decided increase in the amount of urine voided. It is of a very light color, quite limpid, and of low specific gravity, and is sometimes discharged during the convulsive seizure. Digestive disturbances, accompanied by eructations of wind, borborygmi, epigastric pain, and loss of appetite, are present in most cases.

Abstinence from food and continued unconsciousness need hardly be alluded to in this chapter. Cases of this kind derive sensational importance from newspaper description, and from their very hysterical nature suggest fraud and deception. The case of Louise Lateau, as well as others, has been cleverly investigated, and is doubtless familiar to my readers. The history of this class of cases furnishes us with many examples, some of which are quite ancient.

Senneratus<sup>2</sup> writes of three individuals who fasted almost two years, and “yet, though lean, were in good health.”

Upon the authority of Schenck,<sup>3</sup> we are informed that “Katherine Binder, a native of the upper Palatinate in Germany, was said to receive no other nourishment than air for more than nine years. John Caffimer, in the year of our Lord 1585, commanded her to be watched by a Minister of State, Ecclesiastic and two Licentiates in Physic, but they could

<sup>1</sup> Treatise on the Nervous Diseases of Women, London, 1840, p. 229.

<sup>2</sup> Prax Med., p. 212.

<sup>3</sup> Obs. l. 3, p. 306.

make no discovery of her being an impostor, and therefore reported it to be miraculous."

A symptom which I am inclined to think very common, but which is not generally considered so, is the *globus hystericus*. The patient calls attention to a "lump which rises in her throat." It is probably nothing more than a spasmodic contraction of the muscles of the pharynx or oesophagus, or in other cases a morbid, sensory disturbance. It "rises" from the epigastrium, and is attended by dyspnoea and difficulty in deglutition. In some cases obstinate vomiting, which is readily excited by such slight agencies as a hand laid upon the surface of the body, or the administration of a very small amount of food, is a formidable symptom, and unless corrected the patient may become speedily exhausted. In one case which I saw at the request of Dr. Austin Flint, this condition had lasted for several years, and was not relieved by any medication, but was for a time stopped by pressure made over the left ovary.

The disease among males is of interest because of its rarity. A case presented by Bonnemaïson,<sup>1</sup> of Toulouse, may be cited:—

The patient was a man aged 72. The brother of the patient was a hypochondriac; and his mother, who died at the age of 81, suffered from various forms of nervous disturbance, analogous to those of her hysterical son, after reaching her 76th year. The attacks in the case of Dr. Bonnemaïson's patient came on three or four times in the twenty-four hours; ushered in, when occurring during the night, by nightmare; when in the day, by various sensations, and usually by pain in the epigastric region. An aura proceeding from this point traveled along the sternum to the throat, and thence to the mouth and tongue, and other regions of the body, the muscles of the parts affected by this sensation being thrown into violent, rapid, and unaccountable convulsive action. The patient uttered strange cries and yells, or repeated the same words over and over again with extreme rapidity. At times the tongue would be smacked violently against the roof of the mouth, the cheeks spasmodically puffed out with the action of blowing or whistling, and the jaws snapped violently together, without, however, biting the tongue. The arms were moved rhythmically together with the action of flying, or drumming, or playing the piano. Sometimes the lower limbs shook violently, or executed the movements of dancing. The attacks bore a strong resemblance to those of the "convulsionnaires" of St. Médard, or the rhythmic chorea of the epidemics of Louviers, Toulouse, and Morzillac. The disturbance of the voluntary muscles might be accompanied by spasm of the involuntary muscles also, or the latter might form the chief phenomena of the paroxysm, consisting in hiccup, eructations, sighs, and borborygmi. During the whole of the attack the hyperæsthesia of the skin was excessive, especially at the forehead, epigastric region, and sternum; there was no loss of consciousness. The attack ended either with a copious flow of limpid urine, or a discharge of tears. There was never any pain or sensation referable to the generative organs, nor anything whatever in the history of the symptoms indicative of their implication in any way whatever. The same absence of any pathological condition of the organs

<sup>1</sup> Archives Générales de Méd., Jan., 1875. Abst. in Med. News, Oct. 1875.



of generation has been observed in cases of male hysteria observed by others.

Children are not exempt from hysterical troubles, and much of the perversity of young children will often be found to be of this character. If this fact was recognized, a great deal of the suffering in after life might be prevented.

Many of Briquet's cases began before the twelfth year, and it will be found that even before puberty the tendency to this trouble may be often recognized.

Dr. Jacobi,<sup>1</sup> whose careful investigations of the nervous diseases of young children have furnished us with striking facts, looks upon hysteria as an extremely common trouble among young children, connected often with masturbation even in infants of two or three years. Jacobi refers to the tables of Briquet, Amann, and others, to show that hysteria is found frequently before adolescence. Of Amann's cases, 16 of 268 cases were between 8 and 10 years; of those of Althaus—320—seventy-one were before the tenth year. Landouzy collected 300 cases, 48 of whom were between the tenth and fifteenth years.

**Causes.**—Hysteria is most decidedly an affection of women, and is connected in many instances with some sexual or uterine derangement. Among men hysteria is far less rare, I think, than it is supposed to be, but with them the hysterical trouble is of a lighter grade, and it is unusual for examples either of anæsthesia, convulsions, or contractures to be witnessed. As a rule, the hysterical man possesses a smooth face, slender figure, soft falsetto voice, large thyroid cartilages, small hands, and tapering fingers, and sometimes large mammae. His genital organs are poorly developed, and his manners are mincing and effeminate. Hysterical phenomena are, however, not uncommonly presented by stalwart men. Among women this approach to the appearance and behavior of the other sex is inconsistent with the development of hysteria. Women with bushy eyebrows, coarse hair, perhaps a slight moustache, angular build, narrow hips, and coarse voices are seldom hysterical. They are "strong-minded," rarely emotional, and inclined to look upon the hysterical trouble of their weak sisters with something like contempt.

Reynolds aptly says: "Some women are as little likely to become hysterical as some men are to fall pregnant." It might be added: and as their chances to conceive are diminished. Hysteria is of much more common appearance among spinsters and single women, and is far from being rare among old maids who marry late in life. A case of this kind fell under my observation some years ago. An examination revealed an undeveloped uterus; and from the nuptial night dated a series of nervous symptoms of a grave hysterical character. The uterine irritability which is connected with the pregnant state between the ages of thirty and

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<sup>1</sup> On Masturbation and Hysteria in Young Children, by A. Jacobi. *Am. Jour. of Obstetrics, etc.*, vols. viii. and ix., 1876.

forty is apt to produce a profound impression upon the nervous system. Among married women with impotent mates, or among those who have, on the other hand, suffered through the lust, inconsideration, and brutality of husbands of another kind, the disease is not uncommon. The puerperal state, lactation, and the cessation of the catamenia favor its development.

I have lately treated a number of cases of a class which I am sure is familiar to most medical men, especially to those who devote the greater part of their time to the study of nervous disease. I allude to certain ill-defined hysterical conditions that are connected with or follow the puerperal state. These cases do not come under the head of puerperal mania, which is a common and well-recognized form of insanity, but are difficult of description and classification, because of their irregularity. The patients I have seen have all been uræmic at some time during pregnancy, not to the extent which is accompanied by convulsions or other grave symptoms, but the blood-poisoning was much more extensive than it usually is. Barker thinks that albuminuria is not the cause of puerperal mania, but, when found, is merely a coincidence. In the cases I allude to it was *always* present, and seemed to be the cause. I have seen the same symptoms expressed, though in a less marked degree, in patients who were suffering from chronic nephritis, and where the puerperal state had nothing to do with the history.<sup>1</sup>

In the spring of 1875 Mrs. C. came to my office with her husband. I found her to be an amiable, well-educated woman of thirty-two years of age; her manner was cheery and agreeable, and there was no evidence of mental trouble. Three months before this she had been delivered of a child at full term, which was born dead. A week after her milk "dried up." The last months of her pregnancy were attended by evidences of uræmia, marked anasarca, clouded urine excreted in small quantity, but no convulsions or mania. Mrs. C.'s previous history was uneventful. There was absolutely no hereditary predisposition to insanity, and her mind was perfectly clear during pregnancy.

She was anæmic, and complained of dizziness, palpitation, gastric disturbance, vertical headache, loss of memory, ringing in the ears, etc. She passed her urine at the time of her visit in normal amounts, and it did not contain albumen. Her complexion was pale, and her pupils were dilated. A very slight blueness of the skin was apparent, but was confined to the hands. The lips had not lost their lines of expression, which is generally the case in melancholia, and they were not swollen. She was inclined to sleep. Considering that the symptoms indicated "cerebral anæmia," I began with iron, phosphorus, and other remedies of the same kind.

Two days after this visit she again appeared at my office, looking much agitated, and saying that she had come for "protection from herself." She had been tempted to get up from her bed and cut her throat with her husband's razors. She was perfectly cognizant of her condition, and was aware of the fearful nature of the act she was tempted to perform. After a talk of half an hour, she left me, feeling settled, and without the desire.

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<sup>1</sup> Boston Med. and Surg. Journ., June 15, 1876.

On another occasion she came to see me, as "she had the feeling again." She had taken her sister's baby in her lap, and while it was there she "suddenly felt like throwing it on the floor" with all her force. At another time she was prompted to run the blade of a pair of scissors into the fontanelle. These impulses would recur every week or so, when she always came to see me, and would sit a few minutes, talk upon other subjects, and rise to go, saying: "Now, doctor, the feeling has passed off." Not at this time, nor at any other, were there delusions of any kind. Under treatment she improved in general health, and her nervous symptoms disappeared.

Her last morbid impulse occurred during the fourth month after treatment. One evening, with her husband and brother, she went upon the house-top to see a fire. While there the old feeling returned, and she would have thrown herself from the roof, had she not been prevented. This was the last and most serious expression of the disease. Since that time she has not had a return, and says she is perfectly well.

A second case I lately saw was attended by slight though perfectly defined mental changes. The patient was a young married woman of twenty-four years. For some time before parturition and during her pregnancy there was kidney trouble. Before her labor she was a loving and devoted wife, but shortly after lost all of her amiability, and treated her husband and mother with marked coolness, and sometimes with decided rudeness. A month after delivery she took a deep interest in religious matters, and carried the observance of her religious duties to such a pass as to be disagreeable to all about her. She did eccentric things, such as getting up at night, going down to the piano in the drawing-room, and singing hymns. When reminded of the unseasonableness of the hour, she would return to her bed, first shutting the hymn-book in a mechanical manner.

I saw her in this condition, and found a state closely bordering on melancholia, though there was no mental depression, no anxious facies, no sighing, no hopelessness. A persistent use of agents which would restore the action of the kidneys, combined with fresh air and a well-regulated diet, did her much good. After a few weeks the patient slept well, and the mental irritability gradually disappeared.

In both of these cases there were symptoms which were not those of insanity. In Case I. the patient was able to reason, and had full consciousness of her infirmity; so that she had the power to seek the society of others when she felt the impulse. There was the absence of all physical signs of insanity, except the coloration of the skin. In the second case, the short duration of the mental trouble, and its subsidence with improvement of the kidney difficulty, proved it to be a functional derangement.

As regards age, pronounced hysteria rarely begins before the twelfth year; it generally takes its origin at the time of puberty, and from this period may continue through life. It not rarely begins after marriage, or sometimes not until after the menopause, but this is exceptional. In males it begins in middle life, though I have seen the affection among boys. Hysteria is not necessarily a disease of the well-to-do, though indolent habits and luxurious living favor its development; but it frequently appears among



overworked shop-girls who are compelled to stand for many hours during the day. The follies of fashionable life have much to do with the production of a morbid performance of functions of the nervous system. Continued rounds of dissipation, parties and balls which do away with sleep, together with excitement and late suppers, days of idleness spent in reading sensational novels and eating improper food, or tippling liqueurs, especially favor the development of this morbid state. This mode of life, when kept up for some time, especially when the menstrual periods are disregarded, brings about a condition of erethism which expresses itself in the symptoms I have named. Dysmenorrhœa may be attended by attacks, and so may menorrhagia, but many cases occur even when there is no disturbance of menstrual function. Abnormalities of the position of the uterus, and excessive sexual excitement, whether from masturbation or coition, have decided etiological bearing, while warm weather favors the development of attacks. Mental worry, emotional excitement, an attack of illness, and a number of influences of the same kind all act as exciting causes.

**Morbid Anatomy and Pathology.**—Accidental lesions are sometimes found, but so irregular is their character that they are valueless as indications.

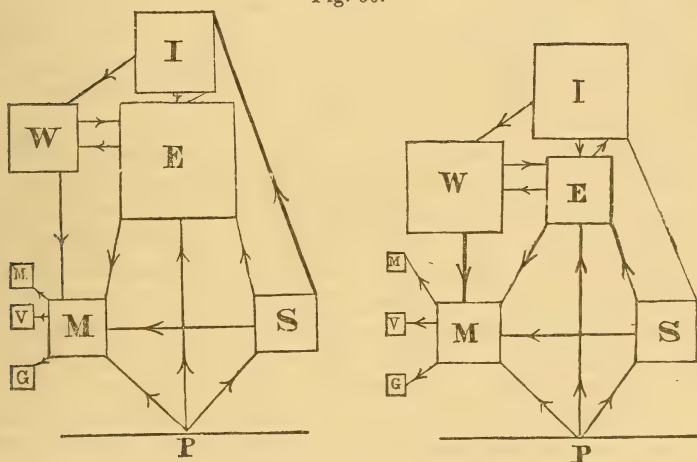
As to the pathology of the affection, very little can be said in addition to what has already been stated in speaking of the symptoms. Hysteria may be said to be a very near relation to insanity, and one writer even considers it a form of insanity; but I should be loath to believe that so many people are actually insane. Hysteria is rather a mental inco-ordination. Emotional exaltation, connected with liveliness of ideation and with feeble volition, and a *paralysis* of judgment, may be said to be the mental condition of an hysterical patient. The balance is lost; and when the emotional side has full play, all the reflex and sensational functions are active and unchecked, while it is only with difficulty that the governing side to which belong volitional and intellectual control is made to counteract the other. This is only brought about by the most powerful agencies, and sometimes *these* are inefficient. If the reader will consult an article by Lauder Brunton,<sup>1</sup> in one of the West Riding Reports, he will find some excellent diagrams which illustrate the mechanism of the nervous centres in the physiology of inhibition.

I have slightly modified the chart of this author by introducing another centre. Let Fig. 60 represent the arrangement of nerve centres concerned in the performance of the functions of the cerebro-spinal system. I. indicates the centre of ideation, E. an emotional centre, W. a will centre, M. a motor centre innervating; *m* (a muscle), *v* (a vessel), and *g* (a gland). S. is a sensory centre, and P. the origin of an external impression. The connecting lines are efferent and afferent nerves. It will be seen that I is in centrifugal communication with W, with M, S, and with E. So that ideas which are evolved without external stimulus may find motor expres-

<sup>1</sup> West Riding Lunatic Asylum Reports, vol. iv. p. 179.

sion either in a voluntary or involuntary manner; may affect the emotional centre, or may be stimulated by impressions received either from that centre or from S. External impressions may be transmitted from P either to S, to E, or to M; in one case being perceived and transmitted to a higher centre, or being converted into a reflex action. E is affected by S and by I, and in turn influences M and I, and to a slight degree W; or on the other hand may be controlled by W. In the normal state we may roughly suppose the proportions of these areas to be represented in the right-hand diagram. In the hysterical state their relative (left-hand diagram) size is greatly altered; E gains in size, and W is very much diminished. The relative size of the communicating tracts also undergoes modification. Though this explanation is decidedly rough and superficial, I trust it will give the reader a better idea of the pathology of this affection than would any extended written description.

Fig. 60.



The Pathology of Hysteria.

**Diagnosis.**—As hysteria may counterfeit nearly every known symptom, it will be seen that the task of making a diagnosis is not always an easy matter. If, however, we consider that the symptoms are generally presented in a group, which is decidedly irregular and its elements inharmonious, and that the patient is on the alert in regard to all that goes on about her; that she has a fear of severe treatment; that the use of chloroform will certainly overcome the contractures; and that the cure is generally sudden, there is not much chance for mistake. Besides, there is never any evidence of gross organic change, the muscles only losing their fulness from inaction. Jannet<sup>1</sup> says that the difference between hysteria and epilepsy, with which it is often confounded, can be detected by the thermometer, there being no change in the former trouble.

<sup>1</sup> De l'hystérie chez l'homme, Thèse de Paris, 1880.

**Prognosis.**—If the individual has suffered for a great length of time, and especially if there be confirmed uterine or ovarian disease, the chances of entire recovery will be extremely bad. The disease is not only discouraging in the way of treatment, but annoying to the friends, and far more disagreeable to the physician, who receives very little for his pains but abuse and want of appreciation. Some cases may be easily cured, and these are among young people. Much, however, depends upon treatment. Dr. Mitchell has known of three deaths from hysteria, and all three were abrupt, and one was due to acute congestion of the kidneys. In two cases that have fallen under my notice, death has taken place in an entirely unexpected way. In one patient there was intense cerebral œdema, and the other, seen by Dr. Ball at my request, rapidly developed uraemic symptoms and died comatose, her death being preceded a few hours by hemiplegia.

**Treatment.**—The history of the treatment of hysteria is curious in the extreme. Going back to the middle ages we find numerous examples of miraculous cures, which were undoubtedly of an hysterical character. Schele de Vere, in his little work entitled “Modern Magic,” thus speaks of a favorite mode of treatment which has been followed by the Zouave Jacob and many others in modern times:—

“The imposition of hands for the purpose of performing miraculous cures has been practised from time immemorial; Chaldees and Brahmins alike using it in cases of malignant disease. The kings of England and of France, and even the counts of Hapsburg in Germany, have been reputed to be able to cure goitres by the touch of their hands. The idea seems to have originated in the high North, King Olave the Saint being reported by Snorre Sturleson as having performed the ceremony. From thence, no doubt, it was carried to England, where the Confessor seems to have been the first to cure goitres.”

“In more recent times a prince, Hohenlohe, in Germany, claimed to have performed many miraculous cures, beginning with Princess Schwarzenberg, whom he commanded in the name of Christ to be well again. Many of his patients, however, were only cured for the moment. When their faith, excited to the utmost, cooled down again, their infirmities returned. Still there remain facts enough in his life to establish the marvellous power of his strong will, when brought to bear upon peculiarly receptive imaginations and aided by earnest prayer.”

Several years ago an individual named Newton went about the country. It was his custom to hire a large hall and extensively advertise. Upon the day appointed he would meet the lame, halt, and blind, and after powerful exhortations and prayers, tell them to form in line and pass one by one before him. The emotional excitement and eager anticipation were sufficient in some instances to divert the hysterical patients who chanced to be among the number, so that in many instances there were spontaneous cures, the lame dropping their crutches, and starting off at a lively gait, and the blind recovering their sight.

Beard, in a paper upon “Mental Therapeutics,” recently called



attention to some experiments he had been making. In many instances of functional disease, he assured the patients that their recovery would take place in some very short time, and found that at the time specified they returned completely cured. This procedure in cases of hysteria is of great value. I have repeatedly stopped an hysterical attack by a douche of cold water or by the exhibition of the cautery. Oftentimes, after the patient has been pleaded with, threatened, and dosed to no effect, a sudden fright or a sharp word or two will do more for her than anything else; but the physician's demeanor to his patient should always be characterized by firmness and dignity, and not by harshness or undue severity.

It is a difficult matter to meet the peculiar manifestation of disordered mental expression in hysteria, for, as we all know, its phases are numerous. No two cases of hysteria are exactly alike, and consequently no two can be treated in the same way. A scolding occasionally does good, as I have just said; but in other cases it would aggravate the patient's condition. We cannot treat the hysterical woman in a trouble-saving and careless way; and though many medical men hold that a sharp word or the direct appeal to the common sense, which is, however, absent here, is all that is required, it will be found that such a course is by no means a wise one to always follow. In many cases it is not best to tell the woman that she is "not to give way," or that she is "not to disgrace herself," for she is unable at once to use her will to overcome all the indirect agencies at work which are acting upon her disordered brain. It is better to gain her confidence, and make her gradually exert her will in new channels by the performance of some act which requires the use of physical force, and this form of exercise may be prescribed by the physician.

As to medication, we may make use of the motor-depressants, bromide of sodium, hyoscyamus; or the mono-bromide of camphor in doses of three grains every hour, till quiet is obtained; the spts. etheris co., chloroform or chloral, and valerian, or its compound, valerianate of zinc. The obstinate vomiting is occasionally stopped by hypodermic injections of morphine; and a belladonna plaster over the irritable ovary will often prove to be an excellent form of treatment. All sources of reflex irritation should be removed as soon as possible, and uterine congestion overcome by leeching the cervix uteri, or hot douches. When there is much irritability of the pelvic organs, I would suggest a combination of tr. cannabis indicus, and bromide of ammonia, with mucilage as a menstruum.

For the anæsthesia and paralysis, strychnia and electricity are the best remedies of which I know, the latter being employed in its induced form, and the electric brush applied upon a dry surface. General treatment of a tonic character should be used when it is possible; and iron, in combination with phosphorus or phosphoric acid, cod-liver oil, and sea-baths, together with local treatment. Local disease should be promptly eradicated if possible, uterine versions or flexions righted, and the menstrual function restored to its regular character. In those bed-ridden cases which are so discouraging and trying, we may use Weir Mitchell's treat-

ment. A patient may lie in bed leading a very irregular life, and doing just about what she chooses, without improving in the least; while, if her room be well lighted, her diet changed, and her muscular tone kept up, a cure may be often wrought.

I am not inclined to place any faith in the wonderful accounts of "metal-therapy" as used in these cases, and in several experiments I have made I have come to the conclusion that the possible increase in sensitiveness came entirely from the warmth of the metal applied or the irritation of the foreign body. If the skin of a perfectly healthy person be subjected to slight rubbing or pressure, and a point be applied, he will feel the application much more acutely than in other parts in the vicinity. For acute paroxysms of hysteria, we may use large enemata containing assafoetida, and if a suppository of this drug in combination with belladonna is inserted every night, a constant influence upon the patient is kept up which is very beneficial.

### HYSTERO-EPILEPSY.

This interesting variety of nervous trouble has received a great deal of attention from Charcot,<sup>1</sup> Dunant,<sup>2</sup> Dubois, and Bourneville, as well as from many other writers, some of whom did not recognize its distinct character until after Charcot's valuable investigations had been announced.

Tissot<sup>3</sup> says that "the hysterical attack sometimes resembles epilepsy, so much so as to have received the name *epileptiform hysteria*, but the attack nevertheless does not possess the true character of epilepsy."

Others, among whom are Briquet,<sup>4</sup> Landouzy, and Saunders, have also described the condition.

Upon the authority of Charcot,<sup>5</sup> the combinations of epilepsy and hysteria take place under the following different circumstances:—

1. *a.* Epilepsy being the primary disease, upon which hysteria is engrafted, under the influence of emotional causes or at the time of puberty.

*b.* After marriage (*vide* Landouzy's Case), the epilepsy having always existed. After connection, the hysterical feature of the attack is developed. In this case the hysterical character of the epilepsy subsided when sexual excitement was interrupted by pregnancy.

2. The hysteria being primary, the epilepsy is added thereto. A rare condition.

3. Convulsive hysteria coexisting with *petit-mal*.

4. An epileptic attack, followed by hysterical contractures, anæsthesia, etc.

I have observed a form which slightly differs from any of the above. The patient, an epileptic, was seized *occasionally* with hystero-epileptic attacks during the menstrual periods, and at other times there was un-

<sup>1</sup> Leçons sur les Maladies du Système Nerveux, part i., Paris, 1872.

<sup>2</sup> De l'Hystéro-épilepsie.

<sup>3</sup> Maladies des Nerfs, quoted by Charcot.

<sup>4</sup> Op. cit.

<sup>5</sup> Op. cit., p. 324.

complicated epilepsy. She has had epilepsy since the fifth year, when she was frightened by her mother, who threatened to beat her.

**Symptoms.**—In an excellent pictorial work published by Bourneville and Regnard, the admirable clinical assistants of Charcot, a number of plates are given, some of which I have reproduced with an abstract of the description by the authors.

“The prodromal features of an hystero-epileptic attack are ovarian hyperæsthesia, the globus hystericus, cardiac palpitation, constriction about the neck, noises in the ears, violent beating of the temporal arteries, obscure vision, etc. The immediate attack is ushered in by irregular respiration, oppression and dyspnœa, awkwardness of speech, amounting to embarrassment, of which the following example, which occurred in one of our author’s cases, may be presented. After the prodromal symptoms described above, the patient, with hesitation and difficulty, enunciated the words: “J’ai . . . l’a . . . respiration . . . dif . . .  
 . . . facile . . . se . . . ne . . . serai . . . pas  
 . . . malade . . . afin . . . de . . . pas . . .  
 avoir . . . de nitrite d’amyle,” in the way they are written. Some tumultuous heaving of the belly then follows, the eyelids palpitate rapidly, the look becomes fixed, the pupils dilated, the gaze is fixed upon some object above, then she loses consciousness.

(Fig. 61.)



TONIC PHASE.—(Bourneville).

The actual attack is characterized by an initial stage (*the tonic phase*) of tonic convulsion. The entire body becomes rigid, the arms being usually stretched out, and the hands are turned in; there is a movement of circumduction of the hands and forearms, the arms being drawn across the body, and the back of the hands brought together, so that the knuckles are approximated (see Fig. 61). The inferior extremities are stretched out, and drawn apart, the feet being in the position of equinus varus, but in other cases the feet may overlap each other, the toes being

<sup>1</sup> See author’s review of Bourneville and Regnard’s work, *Am. Jour. Med. Science*, July, 1879.



strongly flexed. The face is contorted and suffused with blood, and the mouth is often widely opened, or in some cases tightly shut, the lips being compressed over the teeth. Respiration is suspended, the pulse is with difficulty perceived, and the belly is immobile and contracted. The next phase is that characterized by *tetaniform* and *clonic spasms*, the head, which was drawn downwards and to one side, or backwards, returns to its normal position, the facial muscles become seized with clonic spasms, and the eyelids are opened and shut violently but somewhat slowly. A *stertorous phase* supervenes, the face becomes covered with large drops of sweat, the respiration grows noisy and violent, and there is frothing at the mouth. A period of repose then follows, when the respiration appears regular; there are movements of swallowing, abdominal gurglings are heard, and undulations of the abdominal walls become apparent. The *clonic phase*, which has been described as the "stage of contortion," is expressed in two ways, which sometimes succeed each other in the same attack. 1. In clonic movements of the limbs and head, which is rolled from side to side. The face is red and engorged with blood, the neck is stiff, and the arms are stretched out and contracted, and after a time the patient falls violently to the bed, arising and falling again several different times. At the same time the rigidity of the arms disappears, little by little.

2. "The mouth is widely opened, the tongue is protruded; she moves rapidly to the side of the bed crying oh! oh! (*oue! oue!*) The body becomes curved in opisthotonos. She rests on the back of the head and feet, her hair is dishevelled, the legs are convulsed and agitated by alternate movements of flexion and extension." (See Fig 62).

A new period of repose follows.

By far the most interesting phase of the disorder now makes its appearance, viz., the *period of delirium*. In Bourneville's patients, and in fact those of other observers, the incidents of the previous life figure conspicuously in the delirium, and though there is a tendency to the formation of causeless hallucination of the horrible kind, in which reptiles, and such small animals as rats and cats figure at some stage, there is an old impression which serves as a field for the development of a delirium which is exhibited by gesticulations and facial expressions of fear, ecstasy, anger, mockery, erotism, and grief.

The patient at this stage assumes an attitude and expression indicative of her emotional condition. She may remain lying upon the bed, her body inclined to one side, her arms resting by her side, her face upturned and wearing a beseeching look, which constitutes the "Attitude Passionelle" of *Appeal*. At another time she clasps her hands, sits up, turns her face upwards, and gives expression to words of supplication, such as these; "Tu ne veux plus? Encore . . !" this being the "Supplication Amoreuse." At other times the patient lies upon her back, her arms crossed over her breast, and her face wreathed with a most sensuous smile (*erotisme*).

The variations of the delirium do not seem to be at all regular in

their mode of appearance or constancy, but there is a general similarity in the form of emotional excitement and method of expression, and from an inspection of either of the cases, it would appear that for several days at a time there were convulsive attacks followed by delirium, in which scorn, mockery, fear, amorous ecstasy, subsequent repose, and either a return of the delirium, or fresh convulsions, occurred.

(Fig. 62.)



PHASE OF OPISTHOTONOS.—(*Bourneville*).

There may be fifteen or twenty attacks in twenty-four hours, or even many more, and some of these are aborted or irregular, at such times the only manifestations being those of a purely psychical nature; the synopical attacks being examples of this kind. In rare cases the *clonic phase* (or period of the grand movements) is followed directly by the extension of the arms at right angles from the body, so that an appearance is presented which has been called *Crucifiement*, or the position of crucifixion.

This is usually associated with the portrayal of various ecstatic states, which are termed by Bourneville *béatitude*, etc. The first of these is most strikingly portrayed in the plate which is here reproduced. (See Fig. 63).

An occasional feature of one of Bourneville's cases was the complication of chorea, which was manifested at different times in the course of the disease. It was of a rhythmic character, and involved the entire body, so that the trunk was drawn backwards and forwards, the forearms were flexed and extended, the hands were pronated and supinated alternately, and the legs and thighs flexed and extended, the right eyelid became closed, and the muscles of the right side of the neck were convulsed. This occurred in paroxysms, and was modified under ovarian pressure, the movements becoming less violent, and finally ceasing. When the compression was suspended, the movements began anew, and a violent contraction of the right arm and leg, which had lasted during the maintenance of pressure, disappeared. Ether was given, and again the movements were suspended, but a fresh contraction of the limbs of the right side took place.

In one or other of these cases hemianæsthesia and ovarian hyperæsthesia

were observed from time to time. Contraction of various organs was quite frequent, and was sometimes provoked by ovarian pressure, as in the case just detailed, and different visual disorders, such as amaurosis and disordered color sense, were discovered, while hallucinations of vision were prominent in both cases."

(Fig. 63 )

BEATITUDE.—(*Bourneville.*)

The following cases were my own :—

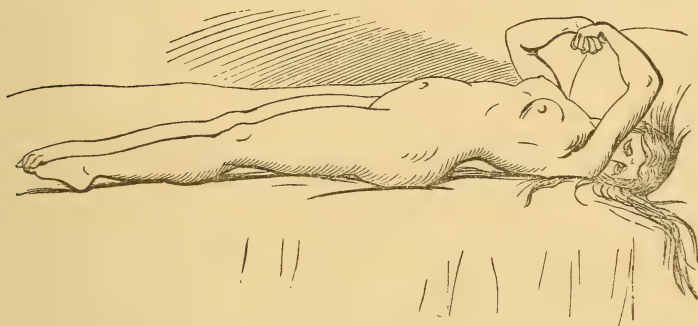
CASE I.—A. P., æt. 18, since the beginning of the menstrual epoch, has suffered from her present form of hystero-epileptic attacks, which have come on generally just after the cessation of the catamenial period. She has been very irregular, and has suffered from amenorrhœa, but there is no uterine disease that I can discover. This amenorrhœa has amounted to an entire cessation of the menstrual flow for several months at a time, during which she would have her attacks. Some of these attacks were like that I shall presently describe, and lasted for several days. There was no succession of attacks, but usually several severe but distinct epileptic seizures, and afterwards an hystero-epileptiform paroxysm. She had been in the Epileptic Hospital for some time, and had given a great deal of trouble by her irritability and mischief-making propensities. Her attacks at the hospital were three in number during one year, each of them lasting from two to three days at a time, during which there was suppression of urine, vomiting, and hemianæsthesia, which in one instance was on the right and twice on the left side.

Her most pronounced attack occurred while she was staying at her mother's house, where I was summoned to see her. This was on the 14th of March, 1877, when her mother came to my office, and told me that her daughter had been ill since the preceding Thursday; that she had



gone with her sister to see a friend; and that while there she had been seized with a severe fit, and could not go home until the next day (March 9). She said that on her return her daughter complained of headache, pain in the back, over the ovaries, and abdominal discomfort, and as the time for her menses had come, she gave her a pill of aloes and myrrh on Saturday, and another on Sunday night, with no result, and a warm hip-bath on Monday. (She had not menstruated since December 1876.) On Monday she had several severe epileptic fits, with frothing at the mouth, during which she bit her tongue, and went to bed, where she remained until I saw her. I went to the house, and found that she had been seemingly unconscious since Monday night, that she had been "frothing at the mouth" since that time, and that on Tuesday she began to mutter and talk to herself; that she had had hallucinations

Fig. 64.



Hystero-Epilepsy.

and delusions, some of them of a painful character, believing that she had been followed by a nurse from the hospital, whose intention was to kill her. When her mother entered the room, she berated her soundly, and was quite abusive, indulging in obscene language.

I found her lying upon the bed, lightly covered by a sheet. The muscles of her back were rigidly contracted, so that her position was one of opisthotonos; her head was turned to one side, and her tongue was protruded. Her eyes were open, and the pupils widely dilated, and insensible to light. Her expression was blank, and she was apparently unmindful of her surroundings. Her arms were drawn over her chest, and her forearms slightly flexed and crossing each other. Her thumbs were bent in, and covered by her other fingers, which were rigidly flexed. Her pulse was 124; temperature,  $101.2^{\circ}$ ; respiration, 33. She was muttering to herself a disconnected string of words without any meaning, and continued them during my visit. She had not eaten for twenty-four hours, and I ordered milk and chloral hydrate in twenty-grain doses, to be forced into her mouth if she did not open it of her own accord.

On my return the next morning, the mother told me that she had had delusions during the night, and had cursed those of her family who ventured to approach her. I found that the rigidity of the previous day had become less marked, but that her right hand and forearm were beneath the lower part of her back. The right corner of her mouth was drawn downwards, and her eyes were still open, and the corneæ anæsthetic.

She did not know me. Temperature 100°; pulse 103; respiration 28. On the following morning Dr. Charles E. Lockwood of this city went with me to see her. She was then much better, and was less rigid, but the right hand was tightly clenched, and no persuasion would induce her to open it. Her toes were also flexed, and her right foot presented the appearance called by Charcot, 'le pied bot hystérique.' Her corneæ were sensitive, and her pupils less dilated. There was some rolling of the eyeballs from side to side, and patient occasionally sighed. Her pulse was now only 96, and was small and irritable; the temperature was 99°. When sharply spoken to, she said "Doctor," and relapsed into a state of stupidity, turning her head from right to left, and staring at the ceiling. She occasionally moved her tongue, as if her mouth was dry. Dr. Lockwood suggested the experiment of frightening her, and so we threatened the use of the cautery, the mention of which first brought forth remonstrance and afterwards a reply to our questions.

Her mother stated that she had not passed urine for several days. I did not find a distended bladder, but when the catheter was introduced, it brought away about half a pint of light-colored urine. This suppression of urine continued for several days.<sup>1</sup> She arose from her bed the day after this last visit, and her menses appeared. During the next three or four days there was slight hemianæsthesia of the right side.

CASE II.—A young lady, 19 years old, had been my patient for nearly a year, during which she had had on an average about one attack of *haut mal* in a week. Her epilepsy dated from the ninth year, and was not dependent upon any discoverable cause. At all times she is irritable, pettish, and techy, and leads a very irregular life. There was nothing remarkable about her attacks; they were not very violent, nor were they connected with any hysterical manifestation. There was rarely any coma; but the attacks were more severe about the time of the menstrual discharge, which was never abundant. On September 12, 1876, I was telegraphed for to see the patient. The day before my arrival, without any premonitions, she had had an attack very much like all the others, but instead of falling asleep she remained convulsed, and apparently unconscious. She vomited two or three times, and became quite cyanotic; so the local physician was sent for. He found it impossible at first to open her mouth to remove the substance which had collected therein and distended the cheeks, and it was only when he was assisted by others that he could do so. She was placed in bed, and remained in this state, the eyeballs rolling from side to side, the body drawn slightly to the right side, and the hands clinched. She became delirious during the night, and had delusions of a lively kind, like those of a patient with delirium tremens. Outbursts of hysterical laughter and jactitations of the limbs followed in the morning, and then she became quiet, but the muscles were somewhat rigid. I arrived at about 2 P. M., and found her lying upon the bed with open eyes and meaningless stare. Her right hand was rigidly abducted, and the bed-clothes were tightly grasped in her hand. The head was drawn so that the chin was approximated somewhat to the chest. The teeth were set together, and there was some grinding of the molars. She breathed noisily, there being an accumulation of mucus in the throat. Temperature 100.2°; pulse 86. The pupils were dilated,

<sup>1</sup> It is probable that this urinary derangement was of the form called by Charcot oliguria.

and seemingly unaffected by light. Pressure upon the right ovary caused her to shrink somewhat. Her abdomen was distended by flatus. During the night she became somewhat relaxed, and muttered unintelligibly, but in a petulant tone. She fell into an apparent sleep about 5 A. M., her respiration being natural. She awoke at about 5 P. M. of the same day (the third), and though somewhat fatigued, arose and went about. She was not hemianæsthetic, but ischuria lasted for several days.

An inspection of the cases of Charcot and others will enable the reader to detect certain symptoms which are alike in all the patients.

CASE III.—Reported by Charcot. Marc —, 23. Hystero-epilepsy dated from the 16th year; attended by hemianæsthesia and hemiparesis of left side. Daltonism of left eye; frequent vomiting. Attack preceded by an aura and pain in left ovary. Attacks included three stages: *a.* Tetaniform contraction, epileptiform convulsions. *b.* Violent movement of trunk and lower extremities (period of contortion). Silly and disconnected talking. Patient appeared to be semi-delirious. *c.* Laughing fits; attacks stopped by ovarian compression.

CASE IV.—Charcot. Cot., 21 years. Hysteria dated from the 15th year, and followed cruel treatment at the hands of her father, when she took to drink and became a prostitute. Local symptoms are: right hemianæsthesia, ovarian pain, permanent, and *tremulation* of the right lower extremity. Convulsions followed ovarian pain; they are tonic, and she bit her tongue and frothed at the mouth. The second period followed at once, and was marked. The attack often terminated by movements of the pelvis, laryngeal constriction, crying attack, passage of large quantities of urine. Ovarian pressure moderated attack, but did not arrest it.

CASE V.—Charcot. Legr. Génévieve, 28. Hysteria dated from puberty. Permanent local symptoms; left hemianæsthesia, ovarian pain, and mental peculiarities (bizarre). Aura quite marked, and so are cardiac palpitation and head symptoms; attack may be divided into three stages: *a.* Epileptiform convulsion, frothing at the mouth, and stertor. *b.* Movement of limbs and body. *c.* Period of delirium, during which she detailed the events of her life. Occasionally last stage would be characterized by hallucinations, when she would see crows, serpents, etc. She would at other times dance. Ovarian pressure arrested attack.

CASE VI.—Charcot. Ler., 48 years. Attacks date from early life, when she was frightened by a dog, and by the sight of the body of a woman who had been assassinated. Local symptoms: hemianæsthesia of ovary; paresis and contractures of the upper and lower right extremities, and occasionally the left. Attacks begin by ovarian aura, followed by epileptiform and tetaniform convulsions, after which she assumed the most trying postures. At the time of the attack she falls into a delirium, during which she indulges in furious invectives, crying to imaginary persons: "Villains, robbers, brigands! fire, fire! Oh the dogs! oh, I'm bitten!" these being suggested by memories of her childish fears. When the convulsive part of the attack is terminated, there follow: 1. Hallucination of sight, the patient seeing skeletons, frightful animals, spectres, etc.; 2. A paralysis of the bladder; 3. A paralysis of the pharynx; 4. Finally, a more or less permanent contracture of the tongue. These last



symptoms remain for several days, during which it is necessary to feed the patient with a stomach pump, and then draw off her urine.

Two cases, reported some years ago,<sup>1</sup> resemble the more modern hystero-epilepsy so closely that I am inclined to infer that they were attacks of this disease.

CASE VII.—Arguinosa's Case. Woman, twenty years. Epileptiform convulsions first showed themselves during infancy, in consequence of head injury. They reappeared at puberty. While residing in the house of Dr. Arguinosa she complained of ovarian pains. The precursory signs of an epileptic attack soon showed themselves, and, on returning from a walk, "she had scarcely time to throw herself on a bed before she lost both sensation and motion. The skin was hot, respiration loud, pupil immovable, eyelids closed convulsively, limbs flexible, while the lips were convulsively moved, or else a sardonic smile sat upon them. Bleeding was about to be practised, when, all of a sudden, after some horripilations, the skin became cold and colorless, the pulse and respiration were suspended, and the patient appeared dead."

Cold affusion to the head seemed to produce an effect. The respiration then became agitated, the pulse strong, and violent convulsions, with tetanic rigidity (pleurosthotonos) set in.

She became angry and irritable, screamed out. Noises in the room, light, and the steps of persons around her were sufficient to "draw her from her attacks of delirium." She had a presentiment of sudden death.

"Two days following there were the same alternatives, the delirium occurring less frequently, and lasting a shorter time; she slept but little that night (the 4th); the next day the only symptoms noticed were aversion to water, light and air, with the pain of stomach previously complained of. On the sixth day she asked for a bath, and the opium which she took in the evening. A stool brought on strong convulsions and noisy delirium. The women who were attending to her believing her to be possessed by the devil, sprinkled her with holy water, which increased her furious cries and bizarre contortions. The following night was dreadful; the mouth full of foam, the eyes injected, and the delirium almost continuous. About ten in the morning immoderate laughter succeeded the previous symptoms. She ultimately died."

CASE VIII.—Ward's case. Mary P., aged 13. Measles at age of 7, and has ever since been subject to cough and pain in the side. About one year ago she had her first epileptic fit, during which she attempted to bite and scratch the bystanders. She was not insensible, but delirious. The attacks came on at intervals for a fortnight afterwards, and they became much worse at the end of this time. Her arms were extended and rigid, and the fingers clenched. At other times she struggled violently, and the abdomen became swelled. She never became unconscious. Her disposition was changed, for she grew exceedingly mischievous between the attacks, developing a propensity for climbing trees and playing the hoyden. Ovarian pain sometimes. The attack is occasionally finished by a fit of laughter.

The so-called *hystero-genic zones* have been described by Richer<sup>2</sup>, Char-

<sup>1</sup> Forbes Winslow's Psychological Journal, vol. ii.

<sup>2</sup> Etudes cliniques sur l'Hystero-epilepsie, etc., Paris, 1881.

cot and Mills<sup>1</sup>, the latter having written a most valuable article upon hystero-epilepsy which will be found to be very complete. These zones consist of limited cutaneous districts which, when subjected to pressure, electric excitation, blistering or hot or cold stimulation, are likely to give rise to, or on the other hand, modify or stop an attack of hystero-epilepsy. These are bi-lateral, and are situated above and below the mammæ, over the ovaries, beneath the axillæ over the ilia, over the seventh cervical spine and the upper dorsal region. The form of excitation varies greatly, whether the patient's surface is or is not hyperæsthetic or anæsthetic, or in proportion to the severity and kind of impression. Occasionally, as has been ascertained, the excitation of these regions during an attack may modify the character of the delusions during the stage of delirium. The so-called *erotogenetic zones* of certain French writers include these as well as other spots—the palmar surface, the back of the neck, and the eyelids—which, when irritated during an attack are followed by changes in the character of the delirium, the patient indulging in erotic fancies.

In simple hysteria, pressure or irritation of these spots may give rise to various dysæsthesiæ.

Charcot holds that a very important diagnostic sign is the reduced temperature. In epilepsy the temperature may even rise to 107.6° F., while that of the hystero-epileptic rarely attains a height of 100° F. In the cases I have alluded to, Case I. presented all the prominent symptoms by him enumerated, and still the temperature was quite high.

**Treatment.**—Nitrite of amyl has been recommended by the French authorities for the suppression of the attack. I would recommend nitroglycerine for the same purpose, in doses of  $\text{m} \text{ v.}$  of the solution spoken of on a previous page. It is of great importance that the pelvic organs be looked after. Dislocation of the ovaries, uterine flexion, or troubles of a like kind, will often be found to have much to do with the genesis of hystero-epilepsy.

## CATALEPSY.

**Definition.**—A disease closely allied to hysteria, of extreme rarity, and characterized by a condition of muscular contraction and semi-rigidity, so that the limbs may be placed in constrained and awkward positions, and remain so for some time. It is attended by loss of consciousness, and cutaneous anæsthesia.

**Symptoms.**—The disease, like epilepsy, is characterized by attacks separated by intervals of greater or less duration, during which periods the patient is usually in apparent good health.

After such prodromata as malaise, vertigo, headache, or functional tremor, the individual will suddenly be seized. He may be talking or eating, when the particular act is arrested, the mouth remaining open, or the hand half raised. The muscles become rigid, but the limb may be moved by the physician or bystander, and if placed in a new position, no matter

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<sup>1</sup> American Journal of Med. Sciences, Oct., 1881, p. 392.

how awkward it will remain so fixed until the muscles are fatigued, when it drops. Individuals are reported to have remained for even an hour or two with legs or arms extended; and in one case I saw the patient remained for half an hour with the right arm extended in a straight line from his shoulder, and the other extended above the head. The position was subsequently changed. The peculiar semi-rigidity of the muscles has gained for it the name *flexibilitas cerea*, on account of a "wax-like" mobility; and there is none of the pronounced stiffness, or, on the other hand, limpness of the limbs, that usually attends the unconscious state. The surface of the body becomes quite cool; the pupils are dilated; respiration is shallow and scarcely perceptible; and it is sometimes difficult to find the pulse, which grows thready, but nevertheless preserves its regularity.

The skin is anæsthetic to an astonishing degree. Needles may be thrust into the tissues without the knowledge of the individual, and pinching, slapping, or other forms of cutaneous stimulation, produce no expression of pain. In a case of hystero-catalepsy, seen with Dr. D. B. St. John Roosa, I repeatedly thrust pins into the arms and legs of a young woman and watched attentively for some sign, but her expression was immobile and tranquil.

It is stated that the electro-muscular contractility is not affected, but reflex excitability seems to be diminished or lost entirely, so that sometimes it is almost impossible to determine whether the patient is alive or dead. The so-called trance states are examples of this kind, and catalepsy has undoubtedly led to burial alive in many instances.

The ordinary attacks usually subside in a few hours, the rigidity growing less marked, and consciousness gradually returning. The attacks, as a rule, follow each other in a series, and then comes an interval of normal health. In this mode of appearance and behaviour, the disease has been likened by Eulenburg to neuralgia. "Strictly speaking, it is rather a cycle of attacks quickly following one another;" and there are remissions characterized by a temporary return of consciousness, and then a fresh relapse, which evidently follows some internal irritation. In rare cases there is a sudden return of consciousness and an ability to perform voluntary acts. The urine and feces are rarely passed in an involuntary manner.

Unless the disease be due to malaria, it becomes chronic, and continues for years. If it is due to malarial poisoning, it usually assumes a regular periodic character, and is amenable to treatment.

**Causes.**—Like many other neuroses, such as hysteria, epilepsy, and those of this class, mental excitement plays no mean part in the etiology of catalepsy. Fright, and other forms of emotional excitement enter into its causation. Injury and malaria may also be mentioned, while masturbation, venery, and intestinal worms are spoken of by writers generally. Jaccoud considers it to be a result or accompaniment of certain forms of melancholia (*Melancholia attonita*), and ecstasy.

It appears as if it were more common in early life, and children are therefore nearly always the victims. Anæmic girls, or boys especially



who study too constantly, are affected more often than those of adult life. Nearly all writers agree that the female is more subject to the disease than the male, and probably the delicate organization of the sexual apparatus has much to do with this. Hereditary influences seem to play a part in the etiology only so far as the general neurotic tendency is concerned. Families in which there is epilepsy, neuralgia, or insanity sometimes include cataleptic members. I have never heard, and I can find no record, of transmitted catalepsy.

**Morbid Anatomy and Pathology.**—Besides the autopsies made by Calmeil and other older writers, which, by the way, throw very little light upon the question of pathology, Schwartz made one autopsy, and Lasegue two, but nothing was found by the latter observer.

Schwartz<sup>1</sup> mentions the case of a boy "who, after an injury, had at first attacks resembling chorea, later cataleptico-tetanic attacks, and after two years died from anæmia and marasmus. There was found in this case, besides a serous effusion in the arachnoid, a softening of the corpus striatum and optic thalamus, on the left side; along the posterior surface of the spinal cord, from the cervical to the lumbar enlargement, was a brownish-red, jelly-like mass, arranged in groups, covering the dura mater. The spinal cord seemed healthy. (There was no microscopic examination.)"

Catalepsy, which is associated with many other interesting perversions of consciousness such as somnambulism, stigmatization, etc., has received a great deal of attention, not only from the laity, but from scientific men of all ages. It is not my purpose to enter extensively into the consideration of these various curious states. The lighter forms, such as the "catalepsie passagère" of Lasegue,<sup>2</sup> have been induced, by mesmerists and others, by passing the hand over the face or body, or by closing the eyelids. The same condition may be induced by looking fixedly at some bright object held close to the face.

A remarkable experiment of a popular nature, which I have repeatedly performed myself, is a curious instance of the susceptibility of certain animals to influences of this kind. If a lobster be placed head downwards, and gentle scratching of the back is made, it will become perfectly quiet, no matter how pugnacious it has been before, and will remain in this position for some time.

The general opinion in regard to the pathology of the affection is that the peculiar muscular condition is due to an increased muscular tone, which probably depends upon impaired voluntary control, so that the muscles respond to trivial irritation reflected upon the spinal ganglion cells.

Volition is checked just as it is in hysteria; and when we consider the theory of "expectant attention," advanced by Carpenter, the genesis of some forms of catalepsy is easily explained. These are the varieties in

<sup>1</sup> Quoted by Eulenburg in Ziemssen's *Encyclopædia*, vol. xiv., translation.

<sup>2</sup> *Archives Gen. de Méd.*, 1865.

which the individual becomes cataleptic when influenced by another. The time has not yet come for the admission of mooted subjects like trance and double consciousness into text-books for students; I therefore await the further development of the subject, which at present is in a chaotic state of confusion.

**Diagnosis.**—The waxy flexibility, which is pathognomonic, is not a feature of any other disease, and this, taken in connection with the loss of consciousness and anæsthesia, makes the diagnosis a matter of certainty. The only point which should interest us is the possibility of simulation. Numerous instances of so-called stigmatization come under this head. There is abundant opportunity for detection, however; and electricity, mental influence, and strong cutaneous revulsives are recommended should we suspect malingering.

**Prognosis.**—When the cause is emotional, or when there is a malarial influence, the individual's chances are remarkably good. It is only when the disease appears in a subject of very marked nervous temperament that there is any reason to give a bad prognosis, and such cases are chronic. A fatal termination is a very remote possibility.

**Treatment.**—Electricity in its induced form seems to be indicated for the abortion or relief of the paroxysm, and amyl nitrite may be recommended for the same purpose. Should there be malarial influences, quinine, arsenic, or iron are of course in order. Curare, bleeding, and many other forms of treatment have been useless. In the transitory affection (*catalepsie passagère*) cold water douches, or diffusible stimulants, are resorted to. The cataleptic and hystero-epileptic conditions are often attended by very great flatus, and when this is removed the patient quite often immediately recovers. An ounce or so of the tincture of assafœtida may be put in a quart of hot water and the woman is to be given an enema therewith, a folded napkin being held by the nurse over the anus. In other cases the rectal tube, such as is used by Emmet, may be tried. I would *strongly discountenance* a modern operation for the removal of the ovaries. I have seen one case where this was tried. The result was death within three or four days. There are so many causes that may enter into the production of catalepsy that it seems an unwarrantable assumption to fix upon the ovaries as the offending organs.<sup>1</sup>

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<sup>1</sup> The Principles and Practice of Gynæcology, 1st Ed., p. 201.

## CHAPTER XV.

## CEREBRO-SPINAL DISEASES (CONTINUED.)

## CHOREA.

**Synonyms.**—St. Vitus's dance; St. John's dance; <sup>1</sup> Paralysis vacillans; Tarantismus; Chorée; Veitz tanz, etc.

**Definition.**—Chorea is a disease characterized by involuntary and disorderly movements of the muscles, is unattended by loss of consciousness and cutaneous sensibility, and may be connected with paresis of certain groups of muscles, or those of one side of the body.

As early as the fifteenth century, a species of religious delusion appeared in Southern and Middle Europe, in an epidemic form, and was connected with certain saltatory and muscular phenomena, which gained for it the name of St. Vitus's dance.

This is described by various writers as a condition of religious excitement characterized by gesticulation, contortions of the body, and leaping, while the patient generally screamed or howled like an animal. This peculiar state was supposed by the older writers to be demoniac possession, and many victims were made to undergo the ordeal, or were put to death by the sword, or burnt at the stake. Under the influence of their condition they sought the shrine of St. Vitus, which was situated in a small chapel near Zabern. Here they were cured by the priests, who sang masses and removed the disorder.<sup>2</sup>

Various epidemics appeared subsequently, but the disease gradually became divested of its noisy character. In Italy a dancing disease, supposed to be due to the bite of the spider, and which received the name of *tarantism*, made its appearance in the early part of the sixteenth century, while at the same time, a peculiar outbreak occurred at Amsterdam, where seventy children of the Orphan Asylum became possessed. They climbed the walls, swallowed needles, hairs, pieces of glass, and other indigestible substances, and "distorted their features and limbs in a fearful manner."<sup>3</sup>

At other places the same thing occurred, and until the end of the seventeenth century, when there was some decrease in superstition, instances of this kind of chronic disorder were common.

<sup>1</sup> For a most entertaining description of this affection read Hecker's *Epidemics of the Middle Ages*, third edition, Sydenham Society's Transactions.

<sup>2</sup> Reynolds's *System of Medicine*, vol. ii.

<sup>3</sup> Schele de Vere's "Modern Magic," p. 357.



**Symptoms.**—The beginning of a simple case of chorea may be the following: The patient, a boy of ten years, who attends school, becomes irritable, loses appetite, and does not care to go out and play with his fellows. He becomes pale and thin, and sits by himself. In a little while some movement of the hand or fingers, some twitching of the face, or dragging of one foot when he walks, attracts the attention of parent or teacher. He may be punished, with the idea that such movements are the result of bad habits or viciousness, but it does no good, and probably increases the trouble. These jactitations cease at night, when he rests uneasily, and is disturbed by bad dreams. This is the condition in which we find the patient. What is the course of the disease? If he is neglected, it will not be long before the convulsive movements become general. The feet may drag along as if paralyzed, and such is the case. He will be unable to button his clothing, or attend to his little wants, and may need the careful and constant attention of his friends. The vocal cords may be affected, and there is as a result a certain aphonia, so that phonation is husky and subdued. Inco-ordination of the lips and tongue gives rise to difficulties in articulation, which are quite distressing, the words being “snapped” and cut short. Mitchell uses the term “habit chorea” for a light form of the trouble, which consists perhaps only of some repeated grimace, or shrugging of the shoulders.

The symptoms are worthy of separate consideration, and we will proceed to discuss them in their order of importance.

1. *Motility*.<sup>1</sup>—The spasms, as I have said, are clonic, and are more often unilateral than bilateral. The right hand is usually affected first, then the leg of the same side may follow, and finally the other side may be implicated, so that the movements are general. The arm is usually involved before the face, though in several of my personal cases the first symptom noticed was a slight twitching about the mouth, and an awkward

<sup>1</sup> In an excellent report of 80 cases of Chorea,\* made by Dr. G. S. Gerhard, of the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases, the following points were observed:—

*Movement.*—In 27 cases, general.

11 “ “ but marked on right side.

10 “ “ “ “ left “

32 “ unilateral, 20 on right, 12 on left side.

In a certain number of these cases the movements shifted to the other side.

*Paralysis.*—Partial paralysis noted in 17 cases. Loss of power in 10 instances confined to right side, in 7 to left.

*Age.*—Under 10 years, 23 cases, 9 m., 19 fem.

From 10 to 20 “ 52 “ 18 “ 34 “

Total, 80 “ 27 “ 53 “

Cure in 56 cases, improvement or “result unknown” in 24 cases.

\* Amer. Journ. of the Medical Sciences.

tendency manifested by the child to open the mouth and draw its breath while speaking. In another, the little boy first attracted the notice of his mother by movements of the alæ of the nose.

I do not think that the movements in chorea are always increased by the effort of the will to stop them, as is the case in sclerosis, in which disease the tremors are exaggerated by any voluntary attempt of the individual at control; and I have often been led to suppose that chorea might be divided into two varieties, viz., one in which the movements are increased with the exercise of the will, the other when they are most violent in a state of rest. The movements of the hands are characteristic, I think. There is a prehensile movement of the fingers and a rubbing of the ball of the thumb and ends of the fingers. There is swinging of the arm, and a shrugging of the shoulder, as if the patient had on large or uncomfortable underclothing.

There is a trivial point which may perhaps be of interest, and I only mention it because it is unique. I allude to the habit which these little patients have of rubbing the seam of the trousers leg by the hand which is affected, for these movements often go on most actively when the arm hangs by the side, and when the attention is not directed to it. In other diseases just such "little straws" will once in a while give a serviceable hint; for instance, in commencing paresis of any kind of the lower limbs. If we examine the tip of the shoe, we will find the sole to be worn down on one side of the body. In locomotor ataxia we will find a reduction of the heel. When these little patients are worried or embarrassed, the movements are greatly increased, and this is one of the strong features of diseases of this kind. I have at present a patient at the Hospital who is almost quiet when in the presence of people he has been associated with for some time, but every new face seems to excite him to such a degree as immediately to give rise to the most violent movements.

The loss of power, which is very often a phenomenon of chorea, is nearly always one-sided, and when it exists to a marked degree, may greatly affect the patient's walk, so that he drags his foot in a helpless manner. Handfield Jones thinks that the want of power is a constant feature of the disease. Such paresis is extremely variable, however, in its extent. Muscular exertion is distressing, and he may not have the power to perform some of the least fatiguing actions of daily life without great prostration.

The muscles that are most paralyzed are always those which have been the seat of the most violent spasm.

*Sensation.*—There may be pain in the wrists if the spasms are severe, or the skin may be anæsthetic; such loss of sensation being confined to the whole paralyzed side, or to a single limb.

*Mental Condition.*—Irritability of temper and emotional excitement are present from the beginning, and the child is restless, sleeps lightly and is tortured by bad dreams. Study or mental application is an impossibility, and spells of crying are quite familiar evidence of the disease,

especially in the earlier stages. Chorea may exist in a very severe form when there is a grave exciting cause; and the convulsive movements may be so violent as to render it necessary to bind or hold the patient in bed. At the request of Dr. J. P. P. White, of New York, I saw with him a case of this kind.

The little girl, who was about ten years of age, had arrived in New York after a sea-voyage, during which the symptoms began. We found her agitated by violent spasms of all four extremities, which had lasted for several days, and it required constant watching to keep her from throwing herself out of bed. They ceased partially during sleep, but this needed repose was denied her to a great extent. Her skin was hot, and her pulse bounding and full. She was perfectly conscious, but complained of pain in the wrists. I inferred, from the general character of the convulsions, their constancy and violence, and from other symptoms, that there was some form of eccentric irritation; and an anthelmintic administered by Dr. White brought away a tapeworm several yards long. The movements disappeared in a very short time.

The urine had been found by Walshe and Bence Jones to be of much higher specific gravity than in health, and to contain an excess of urea. It may vary from 1030 to 1040, and is loaded with the oxalates and lithates.

Another form has been described which is characterized by paroxysms, during which the patient may perform the strangest antics. Her condition before and after the attack is one of quietude, but without warning she becomes agitated by spasms, rolls on the floor, jumps in the air, or rushes about the room. Wood reports a case of this kind, in which the patient, a young married woman who had been slightly ill for some time, developed this paroxysmal variety. "The paroxysms themselves were not always of the same kind. At one time she would be violently and rapidly hurled from side to side in the chair in which she might happen to be sitting, or else, suddenly gaining her feet, she would go on jumping or stamping for a while; or, she would rush around and around the room, and would rap with her hands each article of furniture which lay in her course; or she would spring aloft many times in succession and strike the ceiling with the palm of her hand, so that it became necessary to remove some nails and hooks which had done her an injury; or she would dance upon one leg with the foot of the other leg in her hand."

A professional friend has recently informed me of a case of this kind which came to his knowledge, in which the woman was affected very much in the same way as the patient of Mr. Wood, and that on one occasion she created great commotion by attempting to climb one of the stanchions in the cabin of a steamboat.

These cases are so rare, however, that they only deserve to be mentioned *en passant* as examples of the irregularity of the disease, and are somewhat like the original dances of St. Vitus and St. John.



The following case illustrates a very curious phenomenon of motility which I lately noticed :

The patient, a boy of ten years, was brought to me by his father for treatment, after having been seen by many practitioners, who did not agree in regard to his condition. I saw that his movements were choreic. Questioning revealed the fact that he had never been a strong child, but had always been disposed to nervous troubles ; even the exanthematous fevers, which, like other children, he had had, were generally connected with stupor, and other evidence of susceptibility of the nervous substance to blood-poison. He never had any rheumatic or cardiac affections, and I could hear nothing to indicate valvular trouble. The heart-sounds were sharp and quick, however. Four years ago he began to decline, became weak and anæmic, was irritable, moody, and bad-tempered. His appetite was capricious, and he preferred sweets to other food. In the summer of 1872 the movements in the hands and arms began, and soon became general. His rest was uncomfortable, and he started up in his sleep and cried out. When I saw him four months ago he was a pitiable object. His movements were general. He was unable to hold anything, and was powerless to perform any voluntary actions except those of a gross kind. He could not unbutton his clothing or put on his cap ; his mother even had difficulty in making him walk.

*Variety of Movement.*—Head was violently agitated, there being contractions of the sterno-cleido-mastoideus. He “sucked in his cheeks,” and pursed up his mouth, smacking the lips. Other facial contortions were violent. He winced spasmodically, and there was constant motion of the eyeballs.

*The arms* were in constant motion, but the right was not affected so much as the left. The right arm and hand were slightly paretic, and he was able to force the column of fluid in the fluid dynamometer up to  $16^{\circ}$ , which is equal to 15 lbs. pressure to the square inch. The left forced it up to  $18^{\circ}$ .

*The legs.* The right leg was also slightly paretic. The toe of the shoe was worn down to some degree, although the walk was not noticeably affected.

There was an uneasy rolling of the pelvis when he sat down, and the legs were not entirely under his control. There was pain in the wrists and ankles. Under proper management of his diet he gradually improved, and at the last visit was nearly well. I noticed then for the first time the following peculiar state of affairs. When sitting in front of me, I told him to raise his hands, one after the other. The right hand he raised promptly, but the left he could not, unless he took hold of the wrist with the other hand, and lifted it. This condition struck me as remarkable, especially as he had to repeat the process of aiding with the right hand.

The left hand and forearm might be paretic. There was no loss of electro-muscular contractility, however, but, if anything, it was increased. The muscular power, tested by the dynamometer, was found to be even better than in the other hand. There was no atrophy. With these facts in view, it seemed improbable that this should be the cause.

*It was found that when the other hand was held down, the boy was able to lift his left hand unassisted, and even to raise a dumb-bell weighing 10 lbs., but as soon as the other hand was released he was unable to repeat it.*

To determine whether this was the result of any bad habit, I ascertained from the father that his son had never used one hand to lift the other till a few weeks ago.

In adult life forms of chorea are met with which in nearly every respect resemble those of infancy. Sometimes pregnancy is the cause, and in other cases prolonged emotional excitement, and more especially grief, are in some way connected with the development of the disease.

My case-book contains the records of several of these examples, and their form is usually of that kind which is known as hemichorea, and very often seems to be dependent upon some true organic lesion. In this form the exercise of the will to stop the movements is generally provocative of a decided increase in their violence. The patient is unable to carry food to his mouth, to manage his clothing, or to perform any little acts of necessity. He fears to make any attempts in the presence of other people, and this is especially the case before strangers. I have already alluded to one instance of this kind. In another patient the mere suggestion of meeting a new physician was sufficient to aggravate her convulsive movements.

The chorea occurring during pregnancy generally disappears before parturition, and Jaccoud considers that it may lead to miscarriage, and he has found the mortality greater than in any other form. I am not disposed to agree with him as to the serious character of the disorder.

An instructive case of this disease is subjoined:—

Mary K., æt. 24, entered the Epileptic and Paralytic Hospital July 10th, 1877. She is of nervous temperament, and gives a family history of nervous disease. Her sister has epilepsy, and a brother has infantile paralysis. Up to the fifth day of June, 1877, she was perfectly well. While in bed she was awakened by a storm at about 3 A. M., and was greatly frightened by the loud claps of thunder and the vivid lightning. She arose and fell to the floor, where she lay for some time, crying, but found no difficulty in arising, there being no paralysis. The next day she felt "a cramp" in the left side, and the leg and arm were spasmodically contracted, and afterwards began to twitch. There is no profound loss of power whatever, but some slight paresis of the left side, and a decided hyperæsthesia of this part of the body. The left upper and lower extremities were convulsed by choreiform movements, the hand being more agitated than the leg. The strength of grip is decidedly weakened, and she is only able to force the fluid index in the dynamometer up to 8°, while with the other hand she raised it to 14°. There is some dragging of the foot when she walks. She does not sleep, but requires chloral and other hypnotics. She is in her seventh month of pregnancy, and it was decided not best to try any very active treatment. Arsenic was given, however, in the form of five-minim doses of Fowler's solution, and she became more quiet under its use. At no time has she shown any indication of impending abortion, and though feeble and anæmic, she is able to go about and enjoy herself in a limited way.

*Aug. 25.* Fowler's solution increased, so that she takes  $\text{m. x. t. i. d.}$  Movements somewhat lighter.

*Sept. 20.* Gave birth to a healthy boy after a short labor.

*Oct. 10.* Cured. Discharged. There was no special temperature variations at any time.

A case of interest is that of—

Lena C., æt. 44; Germany; married. Her mother had chorea at the same age. About four years ago, without any appreciable cause, convulsive movements of the whole body began. These were not general at first, and were limited only to the upper extremities. The movements are bilateral, and agitate the hands more than any other part. The facial muscles are slightly affected, and there is a jerking upwards of the corners of the mouth, more especially on the right side. The movements are neither aggravated nor controlled by the will, but cease during sleep. Her cutaneous sensibility is in no way affected, and her sight and hearing are both good. She has a strange habit of clutching her dress in front, probably to steady her hands, and when spoken to she seems greatly disconcerted and moves more than ever.

*June 25.* Fl. ext. conii, ℥ xl, t. i. d. ordered by visiting physician.

*26th.* No marked toxic effects of the drug apparent, except dilatation of the pupils; and the patient says that there is a "complete lightness of the body," and that "she could fly." Some improvement in movements. With a strong voluntary effort the movements are stopped for a time.

*July 10.* Great improvement; patient can hold her arms quite steadily. Discharged at her own request Dec. 15, 1875.

She re-entered Dec. 22, 1875. I found the patient in probably the same state in which she first came into the hospital. She is a spare, tall woman, very restless and emotional. She cannot express herself at all, for when she attempts to speak the tongue refuses to do its part in articulation, and the result is the utterance of ill-arranged sounds, which are not properly formed into words. She smacks her lips, and "clicks" her tongue against the roof of the mouth, and the sounds which come forth are tremulous and agitated, and just such as one would expect to hear from a person who was agitated by some great fear. The contortions of the arms are very violent and irregular, and almost defy description. The body seems to twist upon the pelvis; the arms are thrown backwards and forwards, and the hands and fingers are constantly working. She seems to have no volitional control over her limbs, and has very little muscular force. She walks without any apparent embarrassment, but when seated the movements in the lower extremities are more active than when she stands up. She was somewhat analgesic, as was demonstrated by pinching. Treatment with strychnine considerably moderates the violence of the spasmodic movements.

Chorea may often present a periodic character, especially if malaria enters into its causation. The tendency to relapse is quite a striking feature, and, in many cases which I have seen, it appeared either during the early fall or spring, and reappeared the following season. <sup>1</sup>Weir Mitchell, who has presented some very interesting facts regarding the recurrence of chorea—of 80 cases collected by Dr. Gerhard, 25 had attacks before—some of them several times.

I have two patients now under treatment who have had attacks every spring for the past four years, but in these as well as other cases I find

<sup>1</sup> Treatise upon Diseases of the Nervous System, especially of Women. Phila., 1881.



the disease diminishes in violence, and the attack in duration, as it is repeated. Mitchell has observed cases in which the recurrence of attacks was irregular, a year or two having intervened between them, and such is my experience.

Chorea may be accompanied by other nervous troubles, or exist in an uncomplicated form as a result of debility arising from repeated nervous exhaustion or fresh eccentric causes. In one case I found it to appear as soon as cold weather came, and at the same time an extensive eczema upon the calves of the legs and scalp was developed. This disappeared, together with the movements, under the use of arsenic and oil, but both reappeared the following winter. Dr. E. Frankel has reported a similar case, and I have no doubt there are others who have had a like experience. The disease usually wears itself out in a short time, the tendency to relapse rarely lasting after puberty; and if a cure can be effected, the maintenance of a high standard of general health and certain precautions as to overwork or study prevent a return.

**Causes.**—Various writers agree that the disease is confined to the period between the third and fourteenth years, and this has been my experience. I do not know of a case under three years, but others have seen the disease in younger children. Watson limits the time at which chorea may appear to the period between the first and second dentitions; and Hillier of Great Ormond Street Children's Hospital, has given a table, which is referred to by Radcliffe. He found that of 422 cases at the above institution, 104 were between the ages of ten and twelve. Niemeyer believes the malady to be very rare before the sixth year and after the fifteenth. Girls seem to be more often affected than boys, for what reason I cannot say, except that it may be the more delicate organization of the former, and the preparative changes going on before menstruation.

Mitchell has gone to great trouble to collect statistics showing the influence of season and meteorological changes. He finds that March and April are the two months in which the attacks are more frequent, confirming the observations of other writers; and that the rise and fall of the line of humidity and temperature play a decided aggravating or modifying influence. Mitchell also has ascertained that chorea is very rare among the blacks.

When the disease appears after puberty, it generally takes an eccentric form, or it may be due to central organic changes, or follow hemiplegia. This latter form, denominated by Mitchell post-paralytic chorea, has already been described. In chorea there is a general derangement of the digestive organs and loss of appetite and constipation and palpitation are quite common alterations of function met with in these cases. In the anæmic patients, and they are generally all so, there is often an aortic murmur, and the skin is pale and cool.

The existence of cardiac disease or the previous history of rheumatism is considered by many authors to have much to do with the causation of the disease. Romberg, Hughes, and West, besides many others, have so

decided; and when we consider the pathology of chorea, it will appear to us very reasonable. Of 104 cases of chorea at Guy's Hospital, but 15 of the number were free from any indication of cardiac or rheumatic difficulties.

The disease often follows scarlatina or other zymotic febriculæ, or takes its origin from an attack of acute rheumatism, or whooping-cough. It may result, and generally does, from some directly exciting causes, such as over study, bad air, or food, worms, or sudden fright. My recent investigations in regard to the occurrence of the disease among school children revealed the astounding fact that over twenty per cent. of young school children of the public schools of New York were affected with choreic affections of greater or less gravity.<sup>1</sup> West expresses it as his opinion, that over-study is a common cause, and my investigations are sufficient to prove this.

Many cases are supposed to result from association of unaffected children with those who are the subjects of chorea. Niemeyer alludes to the prevalence of this "mimetic form" among boarding-school pupils. This view has been very popular with the laity, and I am convinced has some importance, still, I cannot but think that the influence of example has been grossly exaggerated.

Malaria seems to play a decided part in the etiology of the disease. This was pointed out by Kinnicutt, who reported some interesting cases in which the movements were aggravated at certain hours on alternate days, and were characterized by something like periodicity.

**Morbid Anatomy and Pathology.**—Comparatively few cases of fatal chorea have been reported. Twenty-two of these are brought forward by Dr. Dickinson, whose excellent article upon the pathology of chorea deserves the attention of every student of neurology. One case has been reported by Ellischer,<sup>2</sup> which is instructive, as it exhibits changes in the nerve-trunks; and Ogle,<sup>3</sup> Kirkes,<sup>4</sup> Hughes,<sup>5</sup> Romberg,<sup>6</sup> and See<sup>7</sup>, have made autopsies in other cases. The connection between disease of the heart and the neurosis under consideration has been studied perhaps most extensively on account of the occurrence of rheumatism and valvular trouble as a complication in many of the cases. In Dickinson's cases the heart was found to be healthy in five; in the remaining seventeen the following lesions were observed:—

|   |        |
|---|--------|
| Recent vegetations on mitral valves only, . . . . . | seven. |
| " " " " " with old thickening, . . . . .            | one.   |

<sup>1</sup> Am. Psychological Journal, Feb. 1876. A number of papers containing questions were sent to the public school teachers of this city. In most instances the answers were intelligent and satisfactory. The cases alluded to above varied from movement of the hands and twitching of the facial muscles to general movements which attracted the attention of visitors.

<sup>2</sup> Archiv. für Path. Anat., etc., Bd. lxi.

<sup>3</sup> Brit. and For. Med.-Chir. Review, January, 1868; Med Times and Gaz., 1866.

<sup>4</sup> London Med. Gazette, 1850; Med. Times and Gaz., 1863.

<sup>5</sup> Guy's Hospital Reports, vol. iv., 1846.

<sup>6</sup> Op. cit.

<sup>7</sup> Referred to by Ziemssen.

|  |      |
|--|------|
| Recent vegetations on mitral and aortic valves, . . . . .                                | one. |
| Recent vegetations on mitral and aortic valves, with pericardial adhesions, . . . . .    | two. |
| Recent vegetations on mitral and tricuspid valves, . . . . .                             | one. |
| Recent vegetations on mitral and tricuspid valves, with pericardial adhesions, . . . . . | one. |
| Recent vegetations on mitral and aortic valves, with recent pericarditis, . . . . .      | two. |
| Recent vegetations on mitral valves, with old pericardial adhesions, . . . . .           | one. |

Of the patients affected with recent endocarditis, the chorea in 6 originated from rheumatism, in 2 from mental causes, in 3 from uterine, in 1 from rheumatic and uterine, in 2 from mental and uterine, and in 3 from unknown causes; thus showing the connection between the rheumatic origin and the cardiac changes.

The brain and cord were affected in 11 cases, there being congestion, softening, and appearances similar to those noted by the other observers I have mentioned.

In one of his cases (No. V.) he made very thorough microscopical examinations, and I present his account of the appearances noted: "Subsequently sections from almost every region of the brain were examined microscopically. They were in most instances natural, the nerve-cells invariably so, save some injection of the vessels, not enough to be decidedly morbid; though the veins were much distended, in particular about the dentate bodies of the cerebellum, the vessels and their canals were normal. There was no extravasation, effusion, or erosion. Two situations, however, were remarkable exceptions to these statements. In the deeper white matter of one of the cerebral convolutions were many conspicuous spots, which consisted of accumulations of crystals of hæmatine mingled with indefinite *débris*, probably of nervous origin, swelling the canals around the arteries which still remained distended with blood.

"The other region referred to as the seat of significant change is that of the corpora striata. These bodies were more minutely injected than the rest of the brain. The capillaries, as well as the larger vessels of both classes, being packed with blood-corpuscles and numerous spots, striking objects under the microscope, were closely set in their substance. These consisted each of an artery in section, empty, crumpled and collapsed, and surrounded by a mass of globular *débris*, which had been formed at the expense of the surrounding tissue. They had evidently been produced by a solution or destruction of tissue around the vessel consequent upon effusion from it, the result of injection which had now ceased to exist. In time these mixed effects of extravasation and disintegration would have disappeared and left mere vacuities.

"The spinal cord displayed loaded vessels and eroded fissures, such as were seen in every other instance examined. In addition to these common changes, the gray matter had undergone extensive transformation of the kind to which the term *sclerosis* has been given. This was slight in the cervical region—extreme throughout the dorsal—absent from the



lumbar. The change was confined to the gray matter, which it affected on the same side of the cord nearly symmetrically. In the dorsal region it involved at least a third of the gray matter as seen in section; the affected portions on each side being adjacent to the attachment of the transverse commissure, and at the root of each posterior *horn*. In the cervical region, though the change was less extensive, its position was the same. The altered gray substance had been converted into a wool-like entanglement of curving areolar fibers, among which nerve-fibers could be sometimes traced, especially near the edges, but from which all other nerve-elements had disappeared, leaving a mere confusion of connective tissue. The nuclei proper to the healthy structure were present, but had undergone no increase, nor was there any other evidence of fibroid or connective new growth. The change seemed to consist essentially of a destruction and removal of the nervous elements, their fibroid skeleton only remaining."

A fatal case of chorea was reported by Dr. Jas. H. Hutchinson.<sup>1</sup> The heart was found affected, the aortic valves incompetent, the leaflets being "swollen and softened," and the aorta was atheromatous above the sinus of Valsalva.

Ellischer,<sup>2</sup> who made an autopsy, found that the vascular changes in the brain were marked, the walls of the vessels being changed, and the surface covered by dark granules. In certain places the calibre of the vessels was narrowed, and there was an accumulation of blood-corpuscles, and consequent effusion of the watery parts of the blood. Some of the vessels contained coagula. The connective tissue about these vessels was thickened and increased in size, and contained yellow pigment and granulated nuclei. The large ganglionic cells in the brain were filled with pigment, and the cell contents much changed. Sections of motor nerves exhibited red patches and destruction of nerve-fibers. These changes show, then, great vascular alteration, and degeneration of normal nerve-tissue.

In regard to the pathology there is much dispute, some observers considering it to be but a functional condition, while others are well satisfied as to its organic nature.

The original observations of Kirkes first demonstrated the relation between chorea and rheumatism. Ogle contends that this relationship (or at least the evidences of rheumatismal causation in the brain, such as emboli) is only demonstrated by fatal cases. He considers the excess of fibrin in the blood to be only the result of the same influence that produces the chorea, and that the blood state, instead of being a cause, may be a consequence of chorea, the result of tissue metamorphosis due to excessive muscular action.

He raises a question as to the disappearance of the movements, and considers this condition of affairs incompatible with organic lesions. This

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<sup>1</sup> Phila. Med. Times, August 5, 1876.

<sup>2</sup> Op. cit.

objection, however, seems to lack force when we remember that in aggravated cases the movements *do not stop during sleep*. Another fact is to be considered, and this is the tendency to relapse which the simplest cases present.

The embolic theory has been advanced by nearly every investigator, and its strongest supporters are Broadbent, Hughlings Jackson, and Bastian. The original investigations of Kirkes served as a basis for this new theory. He found that particles of fibrine were washed into the cerebral vessels. Hughlings Jackson located the place of final deposit in the gray matter of the convolutions in the neighborhood which is supplied by the middle cerebral artery. Jackson very cogently considers the significance of its one-sided character as compared with hemiplegia from embolism, and has since brought up the question of involvement of the muscles more concerned in special voluntary acts, which are likewise conspicuously affected in certain forms of hemiplegia and epilepsy, with cortical degeneration.

Against this theory, some writers have raised the question in regard to the existence of the hemichorea on the same side of the body as that of the brain where the lesion is found, and contend that there must be crossed action. The recent and conclusive investigations of Flechsig alluded to in other parts of this book, show however, that total decussation does not take place in the medulla.

Dupuy and Brown-Séquard have made experiments which prove that such a condition of affairs may exist, and I have myself done the same thing. Since my experiments, I have heard of a case, related by Dr. Walter Hay, of Chicago, in which *post-mortem* examination revealed a cerebral hemorrhage on the side of the hemiplegia.

In one of these experiments made by Dr. F. H. Rankin and myself upon a monkey, electrical irritation (galvanic) of the white matter just beneath the cortex of the upper part of the left ascending parietal convolution produced convulsions in both extremities of the same side.

The views of Jackson now seem to warrant the supposition that in a very large number of cases, in those especially in which no *post-mortem* appearances were found; or at least have not been hitherto looked for in the region of the cortical motor centres where they might have existed unrecognized; that the motor area of the cortex is primarily in fault. In some cases we are furnished with startling proofs of this.

A woman who recently died at the Hospital for Epileptics and Paralytics, and who was in my ward for a number of years, presented the most aggravated symptoms of chorea I have ever seen. Her disease had lasted for twenty or thirty years, and before her death there were decided mental disturbances which occasionally burst out in attacks of mania. Her whole body seemed to be affected, for every limb was agitated by choreic twitchings. She sat usually upon a low chair, her body bent forward, her arms extended, and her fingers spasmodically working. Her head was in a constant state of movement, and her lips and facial muscles were implicated as well. She could not talk distinctly, but her utter-

ances were explosive and rapid. There never had been any paralysis, but after death the important cortical motor centres on both sides were found to be the seat of atrophy. In this case, which probably resembles others of the same class, the destruction of certain psychomotor cortical centres does not result in paralysis, but a loss of governing control upon the part of the upper gray matter, while the lower motor ganglia act independently and inharmoniously in the innervation of the muscular system.

Broadbent localizes the lesions entirely within the corpus striatum. He also calls attention to the existence of peripheral irritation, shock, and various causes which may produce a depraved functional condition.

Bastian adopts the theory that the emboli consist of masses of agglomerated white corpuscles, and that the location of the lesion is in the corpus striatum.

Dickinson is disposed to regard the chorea as the result of rheumatism rather than of endocarditis, and considers the central condition one of hyperæmia of the nervous centres, "not due to any mechanical mischance, but produced by causes mainly of two kinds: one a morbid, probably a humeral, influence which may affect the nervous centres as it affects other organs and tissues; the other, irritation in some mode, usually mental, but sometimes what is called reflex, which especially belongs to and disturbs the nervous system, and affects persons differently according to the inherent mobility of their nature."

In regard to localization he agrees in the main with the other observers. "The spots of perivascular change are widely scattered throughout that large region which lies inferiorly to the cerebral convolutions between the corpora striata and the lower end of the cord; the district of the motor and sensory as distinguished from the mental functions."

It seems, then, that the quality of the lesion is only disputed. I am strongly inclined to accept the embolic theory, not only because the paresis of the limb *may precede any muscular movements*, but because lesions in or about the corpora striata, which produce hemiplegia, may also give rise to choreic movements, but I believe that the motor zone of the cortex is often at first the seat of pathological changes.

**Diagnosis.**—The movements of chorea must be differentiated from those of sclerosis and paralysis agitans. This will not be a difficult task, as the peculiarity of the choreic movement is the *jerk*, while the *tremor* of the other affection is rhythmical and usually *fine*, and varies under certain circumstances. The rapid recovery should also be an element in the diagnosis.

That chorea may result in some secondary disease, such as softening or meningitis, is well settled; and in these cases it will be necessary to take into account the character of *all* the new symptoms, and the history of the old ones.

The exceptional forms of the disease may be mistaken for hysterical troubles, and then the diagnosis will be difficult. It must be borne in mind, however, that this mistake can be made only in adult cases. The



paralysis of chorea may be differentiated from true cerebral or spinal paralysis by its gradual development, and by the age of the individual, as these two forms are quite rare in infancy. Choreic movements usually stop at night, and the exceptions to the rule of quiescence during sleep include those in which the patients have "dreams of movement," such as were alluded to by Marshall Hall.

**Prognosis.**—Chorea is an affection which may very often disappear, without any treatment whatever, in from six weeks to four months; but there are very likely to be relapses. If properly treated, the movements should disappear in from six weeks to two months, or even in a shorter time. If the disease appears after puberty, the prognosis is unfavorable, and all we can do in some cases is to moderate its violence. There is a tendency to recovery in other cases, among them those of pregnancy. Death is a very unusual termination, and it rarely occurs as a result of the disease itself, but rather of some cardiac complication.

**Treatment.**—Internal remedies: Strychnia; arsenic; iron in its various forms (bromide, carbonate, etc.); phosphorus and cod-liver oil. External remedies: Cold to spine—ice, ether spray, and cold douche; Russian or Turkish baths; and salt baths. Rest, diet, and fresh air.

Some of these may be combined with good effect. The plan of treatment I generally employ is the following: Should the child be "run down," as is generally the case, I begin with some preparation of iron, and administer at the same time cod-liver oil. As regards special treatment, I find strychnine serviceable, carried up to the point where stiffness of the sural muscles is arrived at. Next to this stands arsenic. It must be given in large doses; but when we find that digestive troubles are produced very quickly by this drug, strychnia may be substituted. In some cases, when gastritis is produced, we may use the arsenic in the form of Fowler's solution hypodermically, and larger doses may be administered in this way. Cold to the spine cannot be overestimated as a plan of treatment. We may either use the ether spray, which was first suggested for use in this disease by Subetski, of Warsaw, in 1866, or apply ice-bags every day, allowing them to stay on about ten minutes. Perroud, who has used the ether spray, makes applications from four to eight minutes in duration every day. Of thirty-five cases I have treated in this way (I mean with the ether spray), from fifteen to twenty applications produced permanent benefit; and here I would say that the spray should be directed chiefly to the upper part of the cord, over the upper cervical vertebræ. Eserine has been recommended, and Bouchut has given the results of 437 cases, 205 of whom took it in pilular form, and 232 hypodermically. The average dose was from two to five milligrammes. He obtained temporary benefit, which seemed to wear off; but when the drug was repeatedly administered, he accomplished many cures. He reports twenty-three cures by an average of seven injections. It is a dangerous remedy, however, and produces severe gastric symptoms.

The salts of zinc have occasionally proved valuable in cases of this disease; and conium is occasionally efficacious, but its effects are tempora-

ry; but I prefer the remedies I have mentioned. I have found phosphorus, with cod-liver oil, to be a most valuable curative agent, and in cases where everything else failed it has succeeded. This seems reasonable, when we consider how much impaired must be the nutrition of the nervous matter.

Da Costa<sup>1</sup> and Mills,<sup>2</sup> of Philadelphia, have used the bromide of iron; but the latter has had very successful results. In twelve patients to whom he administered the drug, there was no improvement after its use.

Dr. Mills says: "It was usually given in plain syrup and water, commencing with five grains three times daily, as recommended, and rapidly increasing the dose to twenty. The treatment was continued from two to four weeks. Twenty grains very generally caused vomiting. It seems to be a remedy which quickly irritates the intestinal tract."

Oulment and Laurent recommended hyoscyamin in doses of one-sixtieth of a grain, in pill form, at first twice daily, and afterwards more frequently. Amelioration is said to begin in eight or nine days for a child. I have administered hyoscyamin to a number of cases with great benefit. It is, however, a most dangerous remedy, and the commencing dose should not be more than  $\frac{1}{216}$  of a grain, to be increased if dryness of the mouth and dizziness are not too great. Should the presence of worms be suspected, we may either use an injection of quassia and carbolic acid solution (gtt. x—Oj) after each stool, or pursue the ordinary santonine treatment. The use of ferruginous tonics is generally indicated, and those should be selected which are best assimilated and which tax digestion the least. I would therefore recommend either the carbonate of iron, or dialyzed iron. The addition of digitalis seems to increase their good effects quite materially. Chalybeate waters are useful, and sulphur baths are recommended by Baudelocque and others.

Trousseau recommends morphine and strychnine, but I have never seen any good results follow the use of the former; of the virtues of the latter I have already spoken. H. C. Wood recommends a tincture made from the fresh leaves of the skunk-cabbage, with which he has had some success. Electricity I have no faith in, except, perhaps, when the so-called "general electrization" is used as a cutaneous and muscular stimulant. Benedikt has cured many cases by galvanism; but, as far as I can learn, his results are exceptional.

There are instances where nothing does good. It is well to put the patients in a dark room, and keep them perfectly quiet. We will be often astonished at the result. There are little things that must be watched. The diet, above all things, should be regulated with judgment. Plenty of fresh air and sleep come next, and absolute mental rest must be enforced. The school-books and the school-room are to be parted from, and agreeable diversions planned. An excellent auxiliary to our medication is the salt-bath. A handful of rock-salt in the water, and the ener-

<sup>1</sup> Med. and Surg. Reporter, Jan. 30, 1875.

<sup>2</sup> Phila. Med. Times, Sept. 25, 1875.

getic use of the rough towel, will infuse a tone and vigor that will soon become apparent. In conclusion, I must say that decided medication is useless in these patients if their personal habits are not looked after.

### PARALYSIS AGITANS.

**Synonyms.**—Shaking palsy; Parkinson's<sup>1</sup> disease; Trembling palsy; Tremblement sénile; Chorea senile; Chorea festinans.

It is unfortunate that so much confusion exists in regard to the proper classification of this tremor of old age. It has been and is to this day confounded with cerebro-spinal sclerosis.

I shall speak of it as a disease of advanced life, symptomatized by paresis, involving usually the upper extremities, with tremor which is not increased by voluntary muscular action. This tremor rarely affects the muscles of the face, except in advanced stages of the disease, and is accompanied by festination, and in certain cases by bending of the body forwards, and inclination of the chin forwards and downwards.

**Symptoms.**—The extremities first become the seat of tremor, the fingers being agitated in the beginning; the hand is next involved, and afterward the arm. This tremor is bilateral, and it may not make further advances for some time, but ultimately the head, and other limbs are included. The tremor may involve one hand before the other, or the leg of the same side may be next affected, then the leg of the other side, and next the opposite arm. After a variable time, extending from one to ten years, a species of muscular rigidity takes place, so that the head is drawn down, and ultimately the body is bent and the head is thrust forwards, or the chin is drawn down to the breast. The forearms and hands are flexed, and the arms may be drawn to the side of the body. The constant movements may produce an actual abrasion of the skin by friction of the elbows or hands, should the muscular contraction bring them in contact with the body. Any attempt at locomotion is attended by what has been called "festination." The patient may rise slowly from his seat, and perhaps in the early stages walk, slowly though awkwardly, by taking long strides, but when the muscles of the back lose their power, and the body pitches forward, the patient's attempts to preserve his equilibrium result in a shuffling gait, and finally he is compelled to run and gladly clutches the nearest chair or support to avoid falling.

The voice is weak and the speech broken and abrupt, and the form of interruption has been compared by Charcot "to that which affects a novice in equitation when his horse begins to trot." This interruption is caused by the violence of the muscular movements. The patient pitches his voice when he begins to speak, and never changes the tone until he has finished, so that his phonation is decidedly monotonous. He is greatly fatigued by the constant muscular movements, and is restless and inclined to seek new positions which may give him ease. A disagreeable symptom is the occurrence of cramps of temporary duration, which are more com-

<sup>1</sup> Essay on Shaking Palsy, London, 1817.



mon during the day. During the tremor the fingers or toes may be rigidly flexed or extended. The face is utterly devoid of expression, but the mind is never impaired, and there are no affections of the organs of special sense. The tremor in the beginning ceases at night, but in the established form it is present at all times.

The termination of the disease may be in death through exhaustion or complicating diseases, such as pneumonia, which carried off three cases reported by Trousseau. The functions of the bladder and rectum are not usually involved, except when the disease has become confirmed. In one case Topinard found sugar in the urine, but it is hardly necessary to say that this circumstance is exceptional.

After suffering for a number of years the patient is finally obliged to seek his bed, sloughs form over the sacrum, and he gradually sinks, the tremor, perhaps, moderating slightly before death.

The following interesting case is one that illustrates the course of the disease perfectly :—

Mr. M., the patient, during his early years led an active life, and after following the occupation of a peddler gradually worked his way up to prosperity. For years he went about the streets of New York carrying, many hours in the day, a heavy pack upon his back, and during this time he suffered many privations of food, rest, and sleep, and was exposed to the elements, after going home wet and cold. About fifteen years ago he first noticed the appearance of his present disease. He is a stout man of large frame, and about 70 years old. The trembling began after slight exertion, and continued for some time. It became more pronounced and constant during the next two or three years, and he was unable to unbutton his clothing, feed himself, or use his hands. His general health did not seemingly suffer, but he was "nervous" and depressed, and fully aware of his pitiable state. He did not tremble so much when lying down, but when he moved about or assumed the erect position the hands shook and the head shook constantly from side to side. The movements always stopped at night, but it was some time before he could sleep. He gradually lost power; the right arm losing strength primarily, and afterwards the left. Coincident with the loss of power there was tremor. When I saw him two years ago, I found him seated in a chair in which he had difficulty in keeping his place. His upper extremities and head were chiefly affected. The head was inclined forwards, and was constantly agitated by movements of a rhythmical character, which did not appear to be increased or diminished by any act of volition. He could not raise his chin, but looked up at me when I entered the room with his son. When asked a question, he answered in a tremulous voice, speaking as would one who was chilled. His body was curved forwards, and his arms were semi-flexed, the elbows being drawn to the chest; and forcible or voluntary extension was impossible. There was no atrophy of the muscles of the arms or forearms, and no decided loss of sensation. The hands were agitated by the same rhythmical tremors as the head. When he was lifted up he could not walk, and would have pitched forward if not held. In this position I noticed that the knees were also affected by the tremor. His bladder and rectum did not seem to be involved, at least not as a result of the disease, for beyond symptoms of enlarged prostate he suffered no impairment of function. For the past two years he has needed powerful

opiates to procure sleep, the movements continuing unless they are given. He swallows with difficulty, and there is a drain of saliva from the corner of his mouth. As far as I can learn there have been no disorders of the organs of special sense, and certainly there are now none. His mind seems to be somewhat affected, as he is irritable and silly, and his memory is deficient.

It may be stated that the affection may exist in a modified form (Parkinson's disease) and that tremor alone may be the only symptom. Festination and rigidity are by no means constant expressions of the affection.

**Causes.**—Nothing is known in regard to the causes of paralysis agitans. It has followed mental distress, or has been preceded by neuralgia and rheumatism, but these seem to be connected with so many nervous diseases that it is difficult to say just how much they have to do with the etiology of paralysis agitans. I have seen several cases, and in none of them was there any history of predisposing or exciting causes. We know that the disease is rare before the fortieth year, and that the male sex is more often affected than the other sex.

**Morbid Anatomy and Pathology.**—Handfield Jones<sup>1</sup> holds to the doctrine that the affection is purely of a functional character while others believe it to be a multiple cerebral sclerosis. In an excellent review of the recent writings of Charcot and Moxon, which has appeared lately, the reviewer says: "There is a certain satiric humor in Professor Charcot's notice of the morbid anatomy of paralysis agitans. He divides the autopsies hitherto made into three groups. In the first group nothing at all was found. The second group comprises cases of supposed paralysis agitans, which Prof. Charcot considers were in reality sclerosis; and the third group contains the case of Parkinson subsequently mentioned, and a similar case by Oppolzer, which is treated with similar distrust. There are, however, other cases on record which give much more satisfactory results. Leyden has reported one in which the agitation was limited to the right arm, and a sarcoma the size of a large nut was found in the optic thalamus of the opposite side. Murchison and Cayley have reported a case in which very definite changes, partly of sclerosis and partly of cell growth, were found in the cord; but as in this case the symptoms are described but very briefly, it is possible that Prof. Charcot would place it in his second group. Joffroy, however, took especial care to investigate this point, as to whether the cases were really paralysis agitans or insular sclerosis, and he states that two out of his three cases were clearly paralysis agitans. In these two cases there was exuberant growth of the epithelium of the central canal and of the nuclei around. In the third case, which seems not to have been a very doubtful one, there was in addition a sclerosed patch in the medulla."<sup>2</sup>

The pathology of tremor is still so imperfectly understood, and there is

<sup>1</sup> Functional Nervous Diseases, p. 382.

<sup>2</sup> Brit. and For. Med.-Chir. Rev., Oct. 1875.

so much to be said, that it would involve a much more protracted consideration than the size of this book will permit. We may, however, consider some of the physiological conditions of muscles which, when disturbed, result in the pathological state known as tremor.

The variation or interruption of any compound entity is followed by an inharmonious relation of its parts; thus a musical sound is the result of a number of more or less rapid vibrations and waves, their number influencing pitch. If a catgut string in a state of tension is twanged, vibrations are induced and a musical tone is produced; but if a stick be loosely held against the string, without actual pressure being made, the vibrations will be interrupted, and a discordant noise will be the result of such contact. It has been demonstrated that a visible muscular contraction is, after all, the result of an incredible number of smaller contractions, which cannot be seen with the naked eye, but may easily be appreciated with the aid of the myographium or some other registering instrument. Upon faradizing a muscle this may be experimentally demonstrated. Shorts breaks are followed by visible contractions of the muscle and movements of the limb; but if by a proper current-breaker this interruption be repeated many hundred times a minute, the intervals will be so short that, though an immense number of rapid contractions take place, there is but one grand contraction of the muscle which is appreciable.

In the physiological state this co-ordination (if I may use the word) of the minor contractions is so perfect that the muscular movements are steady and separated by regular intervals; but when the rhythm is lost, or the harmony destroyed, the smaller contractions will be separated by intervals of sufficient length to be seen, and tremor results, the degree of tremor being proportionate to the length of the interval.

The filaments of a tired muscle, the motor centres being worn out, do not contract evenly; so, as a consequence, there is a visible tremulousness. In functional tremor, such as characterizes the disease in question, this is undoubtedly the pathological condition.

**Diagnosis.**—The treatment of cerebro-spinal sclerosis may be mistaken for that of paralysis agitans. Let us compare the points of difference:—

## PARALYSIS AGITANS.

Tremor continues, but not increased by voluntary efforts.

Tremor regular and "fine."

Facial muscles unaffected.

Runs forward to preserve balance.

Speech slow, or affected by violence of muscular movements.

A disease of old age, or advanced life.

## CEREBRO-SPINAL SCLEROSIS.

Tremor subsides during repose, and is always aggravated by volitional attempts at control.

Tremor "coarse."

Usually cranial nerve paralysis, or tremor of facial muscles.

Only staggers when walking is attempted.

Speech-defects those which arise from paralysis.

Usually a disease which appears before middle age.



Mercurial tremor, lead tremor, and alcoholic tremor sometimes resemble that of the disease in question; the former is, however, more violent in the morning; the tremor from lead is attended usually by colic and other symptoms of plumbism; while no doubt need arise in regard to the third, which is attended by evidences of alcoholism. Post-paralytic chorea may be excluded by the history of hemiplegia or some other equally prominent organic condition, and the tremor is aggravated by voluntary efforts. A functional tremor of a very light grade, which is simply a personal peculiarity, is met with sometimes, and should not be magnified to the dignity of a disease. This may affect several members of the same family, as is the case in one example of which I know. The head of the family is a vestryman of a church, and in passing the plate he sometimes is obliged to exercise the utmost self-control to prevent the contents from being thrown out, and more than once this infirmity has given rise to insinuations concerning his habits. His two children, both very young and healthy people, are affected by the same tremor. In such a case the trouble does not increase with time, and there are none of the other progressive signs of the true affection.

**Prognosis.**—The course of paralysis agitans is decidedly progressive, though very gradual, and the individual may live for ten, twenty, or even thirty years after the appearance of the tremor. When death takes place, it is in nine cases out of ten the result of some other disease. I am convinced that *genuine* paralysis agitans is never cured, though it may be relieved; and it is highly important to distinguish simple functional tremor, which is not uncommon, from the disease under consideration. This functional disorder is amenable to treatment.

**Treatment.**—Handfield Jones<sup>1</sup> considers that nothing can be done for the disease among very old people when it has become decidedly chronic. He has used electricity, conium, and a variety of remedies. "The general tenor of experience in this and in kindred disorders is to the effect: (1) that the main indication is to nourish and support the failing power of the nervous centres affected; (2) that this is best accomplished by remedies drawn from the class of sedatives, or by the milder tonics. Henbane, conium, chloral, subcutaneous opiates, bromide of potassium, belladonna, hypophosphites, or phosphorus, cod-liver oil, carbonate of iron, and sulphuret of potassium baths, with electricity in one or other of its three forms, appear to me the most hopeful remedies. But steady persistence in appropriate treatment is doubtless essential, and the want of this may account for many failures. Trousseau's adage should be borne in mind, '*A longue maladie, longue traitement.*'"

He refers to a cure reported by another observer. The patient was a woman, eighty years old, in whom the disease followed severe labor; and she was ultimately unable to carry trays or heavy loads. The faradic current used several times effected the disappearance of the tremor. I am

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<sup>1</sup> Brit. Med. Journal, March 8, 1873.

inclined, however, to consider this case one of functional tremor, and not of the grave variety I have described.

I have used conium with good results, and find that it relieves the patient, but after the use of the drug has been discontinued for a few weeks, the tremor is pretty sure to reappear. It should be given in doses of the fluid extract of from  $\mathfrak{m}$  v— $\mathfrak{m}$  viij thrice daily.

Hyoscyamin, a remedy that possesses virtues second to none as a depresso-motor, is worthy of a trial in this affection, although in chronic cases its good effects are rarely more than temporary.

Elliotson<sup>1</sup> has cured a case by the carbonate of iron in large doses, and strychnine has been suggested, but it is doubtful whether it does any real good.

Galvanization of the spine, one pole placed over the spine, and the other as near as possible to the point of exit of the spinal nerves, has been advised; and in some instances it has improved, if it has not cured, the affection.

## EXOPHTHALMIC GOITRE.

**Synonyms.**—Basedow's disease; Graves' disease; Exophthalmic cachectique; *Cardiognus strumosus*.

This interesting disease has received but little attention until within a few years, and it is only lately that it has been considered as a neurosis.

**Definition.**—Exophthalmic goitre is a disease connected with vascular excitement and circulatory disturbance; there is not only enlargement of the thyroid gland, but an excessive engorgement of the intra-orbital vessels, so that the eyeballs are pressed forward, giving rise to a hideous deformity.

**Symptoms.**—The first symptoms of the disease are generally indicated by violent action of the heart, and great acceleration in the circulation; and with this there is hyperæmia of the cerebral vessels. Palpitation and pain over the left side of the chest, shortness of breath, and flushing of the face are other symptoms of this early stage. This early vascular disturbance is, perhaps, the first evidence of the disease noticed by the patient, but the enlargement of the thyroid gland may have been progressing for some time. There may be other early symptoms which appear with increased growth of the goitre, and protrusion of the eyeballs. These are falling out of the hair of the eyebrows, as well as the eyelashes.

The heart's action is violent throughout the disease, and the pulse may beat from 120 to 140 per minute; while the temperature is one or two degrees higher than the normal standard. There is nearly always a systolic bruit and a carotid murmur. The hand, when placed over the goitre, may receive a peculiar sensation, which is produced by the agitation of the thyroid by the rapidly circulating blood in the enlarged vessels.

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<sup>1</sup> Quoted by Jaccoud, *op. cit.*, vol. i., p. 427.

Although the disease begins suddenly in some instances, it is usually of slow development, and, according to Eulenburg, there may be hysterical manifestations before the pulse acceleration manifests itself. I have myself noticed that the patients then seen were emotional and easily excited.

Digestion is nearly always impaired, and there may be some diarrhœa or attacks of vomiting; while sleep is troubled, and the patient suffers greatly for want of rest. His appearance is unmistakable. One or both eyes are prominent, and uncovered by the lids; and the sclerotic is exposed above the cornea to a great extent. The patient is hypermetropic, and suffers considerably from conjunctivitis produced by the irritation of foreign bodies which lodge there.

There is rarely any visual disturbance, although troubles of accommodation are met with; and there are no changes to be observed in the retina.

Dr. Yeo reports two very valuable cases, which are presented in admirable shape in a late number of the *British Medical Journal*.<sup>1</sup> In one of these there was exophthalmos of the left eye only, the goitre being on the right side. The second case was thus described by Dr. Yeo: "The patient is a young single woman, 23 years of age, robust and strong-looking. She shows no signs of the pronounced cachexia (phthisical) so evident in the other patient. But she is especially interesting now, as being also the subject of unilateral exophthalmos. In her case the right eye only is prominent. There is very little, if any, enlargement of the thyroid, but there is constant palpitation. The pulse has varied during the time she has been under observation from 116 to 140. She comes of a healthy family, and has always had good health till lately. She first noticed the prominence of the right eye about a year ago. All this time she has been feeling nervous and excitable. She came to King's College Hospital about nine months ago complaining of pains in the back of the head and palpitation. She stated, also, that she suffered frequently from 'bilious attacks,' attacks of vomiting which would last a whole day, after which her throat would get very large. She complained, also, of frequent profuse perspirations coming on twice and three times a day, sometimes without any cause and sometimes on the slightest exertion. The hands and feet are always perspiring, and her hair is sometimes wringing wet." She is easily fatigued, has lost her appetite, and is much thinner than she used to be. She suffers much from dysmenorrhœa, and all her symptoms are worse at her periods. She says her throat was much more enlarged nine months ago than it is now.

There may be double exophthalmos or single, but the double affection of the eyes is the rule in the great proportion of cases. In some cases it is absent entirely, and of 58 cases reported by Von Dusch it was absent in four.

The eyeball may be pressed back, as the vascular cushion behind is

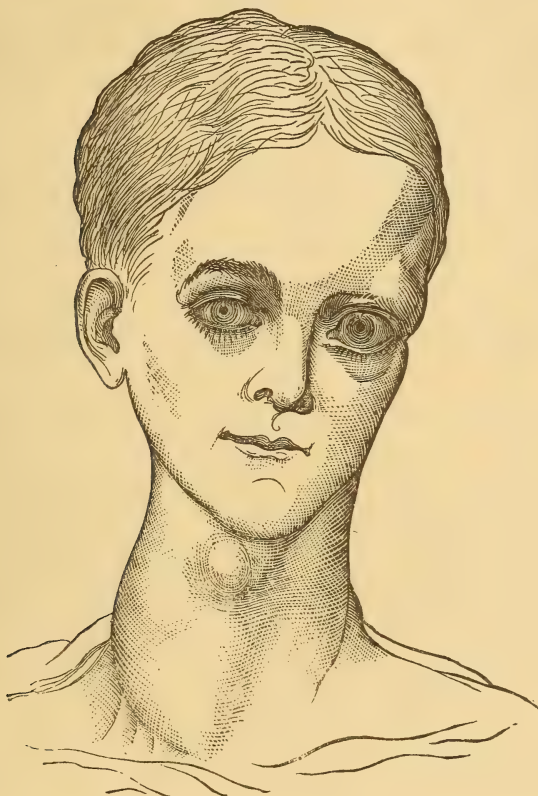
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<sup>1</sup> March 17, 1877.



soft and yielding; and a peculiar thrill is felt. An "arcus senilis" has repeatedly been observed by Bartholow,<sup>1</sup> who first called attention to this change, and by others afterward, among them Thomas.<sup>2</sup> Von Graefe was the first to allude to the peculiar behavior of the upper lid,

Fig. 65.



Dr. Yeo's Case of Exophthalmic Goitre.

which, as Eulenburg expresses it, "loses its power to move in harmony with the eyeball in the act of looking up or down." Irritability of temper, hysteria, laryngeal trouble, and difficulty of breathing are symptoms which are to be noticed, and towards the end this respiratory embarrassment becomes quite distressing.

The patient is generally badly nourished, and we may have added to the symptoms already described, many of those of general anæmia.

The skin of the whole body may sometimes be of a much darker hue than it is in a condition of health, and some discoloration of that covering

<sup>1</sup> Chicago Journal of Nervous and Mental Diseases, July, 1875.

<sup>2</sup> Richmond and Louisville Med. Journ., Nov. 1876.

the forehead is often noticed. This discoloration resembles a brown stain, and it has been spoken of as "bronze skin" by some writers. Raynaud<sup>1</sup> has called attention to the connection between this stain, or vitiligo, and exophthalmic goitre. He gives "five cases of exophthalmic goitre, culled from various sources, in the course of which patches of vitiligo appeared on various parts of the body. Beyond the observation that vitiligo is more common in men than in women, except when congenital, that it attacks by preference persons of dark complexion, that it is sometimes, though rarely, hereditary, and has a certain analogy to Addison's disease, viewed as an imperfect vitiligo, little has been made out with regard to its pathology. Mr. Hutchinson has pointed out that although no known cachexia appears to set up a predisposition to the affection, the symmetry of the cutaneous patches is suggestive of some pre-existing general fault of the circulatory or nervous systems, and is opposed to the hypothesis of a parasitic origin. Without offering any explanation of the coexistence of vitiligo with exophthalmic goitre, Dr. Raynaud thinks that the coincidence should not be allowed to pass unnoticed."

The connection of urticaria has been pointed out by Bulkley, who reports two cases of the disease. One of these is presented:—

"Mrs. —, aged 45, was delicate and sickly when a child. Was married at 18 years of age, but separated from her husband after 4 months; she had a miscarriage at 3 months, and has never been completely well since. She is of full habit; bowels and menses regular; tongue coated; pulse 84, weak; has had chronic rheumatism.

"The history of the Graves' disease dates back a number of years—at least five years previous to my seeing her. This diagnosis was made by a prominent oculist whom she consulted about the projection of her left eye. She has been treated much of the time ineffectually by various physicians, remaining with each long enough only to experience more or less benefit, and then changing. The eyes exhibit clearly the peculiar appearance of patients with exophthalmic goitre, the left one being more strikingly prominent, and being of but little service for vision, she soon losing control of it. The other phenomena of the disease have been present for some years—irregularity of the heart's action, and at times severe palpitation, and enlargement of the thyroid; but this is not so very marked.

"Five years before coming to me she experienced a severe nervous shock, and dates her skin trouble from that period. She states that she has not perspired since. She began then to have 'a fine rash and redness all over the body,' and itching. This continued about the same, off and on, for four years, when, after being weak and exhausted, and having various hysterical difficulties, the itching became more general, and an eruption corresponding to that now existing appeared. Lumps would form on the forehead and on various parts of the body; sometimes the face and head would appear greatly swollen.

"When first seen she was in a pitiable state of nervous anxiety; the itching of the feet and toes and sometimes of other parts of the body she

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<sup>1</sup> Archives Gén., June, 1875; and London Med. Record, Sept. 15, 1875.

described as agony. At the first visit there was not so much to be seen on the skin, but there were a few urticarial blotches on various parts of the body and limbs. While under observation, however, she had several acute attacks of skin trouble, all of the same sort. On one occasion she woke with the upper lip greatly swollen, and with swellings on various parts of the body. On the following day, when seen, the whole face was swollen and puffy; on the middle of the forehead there was a large erythematous lump, also one beneath the right eye, and smaller ones about the face. The hands were swollen; on the right hand, near the little finger, there was an erythematous patch, somewhat swollen and with two small vesicles on it. There were also various erythematous and urticarial blotches about both hands and wrists; and on the back of the left hand, near the thumb, there was a red spot with the skin broken, as if the seat of a former vesicle. The whole surface of the skin burned as if scalded or scratched; there was no pain on deep pressure. On another occasion, a day or two after there had been, according to her statement, numerous swellings on various parts of the body, the remains of several were visible on the right cheek, and on the arms there were numerous stains, some of them quite dark, as if the parts had been bruised—the remains of the lumps; the hands and arms were manifestly swollen, and there were urticarial wheals on the limbs and body.”

The following case is one of unilateral thyroid enlargement, with double exophthalmos:—

Mrs. L. B., 28, U. S.; milliner. Was always well until eight years ago, when her present difficulty began. She was then living in New York, and actively employed. At this time she noticed the growth of a goitre upon the right side of the neck, which pulsated violently when she was excited or over-fatigued. She then flushed easily, and often had headaches, which were quite intense. These she has now, and her pain is of the congestive variety, and diffused. She presented herself at the out-patient department of the New York Hospital, complaining of a pain just beneath the border of the last rib on the left side, which was quite constant, but not increased by pressure, or by taking a long breath, or after eating. The pain was most severe in the morning, and seemed to move off towards night. Her heart seemed healthy, so far as valvular lesions were concerned, for no abnormal murmur was present; but there was great rapidity of action, the pulse-beats varying from 106–120 per minute. The pulse was also quite bounding, and full. The carotids pulsated quite strongly, and there was a very marked venous thrill perceptible in the jugulars. Upon the right side of the neck, just above the sterno-clavicular articulation, and extending laterally, there was a tumor measuring  $2\frac{1}{2}$  inches in length, and about 2 inches in breadth. The marked pulsation of this growth led Dr. Slaughter and myself to suppose at first that it was an aneurism, but we were unable to reduce it by pressure, or to diminish its size by compression of the carotid; and there was no history of injury. The peculiar movement was due to the pulsation of the carotid upon which it rested above, and laterally passed the right jugular vein, which was also agitated by the transmitted pulsation of the carotid. When the hand was placed upon the enlargement there was perceived an undulatory or “purring” movement. No bruit was heard with the stethoscope, but the tracheal sound was readily perceived. This growth under-



went variation in its size. Cold weather seemed to influence it in this way, and stimulants, or other agencies which increased the blood pressure, materially modified its size. The face was puffed, bloated, and red, and the eyeballs were somewhat prominent, while the pupils were dilated, and the irides rather sluggish. She was not hypermetropic, and there were no other defects noticed. By steady pressure I was enabled to perceive the "cushion feeling" alluded to by medical writers who have observed this disease. Her companions twitted her in regard to her fixed stare, which resulted from the exophthalmos. Her ankles and feet were œdematous, and pitted deeply on pressure. Her urinary organs seemed to be in order, and there were no indications of renal disease. She has noticed at times patches of rusty discoloration which appeared about her neck and upon the left side of her face. These lasted for several days, and then faded away. She has had several minor symptoms, such as nose-bleed, which occurs even now, every two or three weeks. Her menses are scant, but there is apparently no uterine disease. Her digestion is feeble, and she is slightly constipated. R.—Ext. ergotæ fl. ʒj, t. i. d.

**Causes.**—The disease is one of adult life, and there are about twice as many females as males affected. But few cases have been reported in which the disease appeared before puberty. Devol saw a case, the patient being a girl of two and one-half years. It is connected, in some cases, with metrorrhagia, or hæmorrhoidal bleeding, or in others with heart disease; but though many authors consider anæmia to be an important cause, others are doubtful.

Examples of traumatic origin have been noted by Begbie<sup>1</sup> and Von Graefe,<sup>2</sup> and others have been apparently of idiopathic origin. The case of the first followed injury to the occiput.

**Morbid Anatomy and Pathology.**—The observations of those who have made autopsies, differ greatly. Morel Mackenzie found softening of the corpora quadrigemina and the posterior part of the medulla. The heart was not much affected, there being only slight atheromatous deposits on the mitral and aortic valves, with thinness. Other observers have found hypertrophy of the heart and insufficiency of its valves, but in other cases there were no heart lesions whatever. The thyroid gland has been found to contain enlarged vessels, and the orbits an increased quantity of fatty tissue. In one of Begbie's cases there was sinking of the eyeballs in the orbital cavities after death.

Much discussion has taken place in regard to the pathology of the affection, but recent investigations point to the nervous origin of the disease. The cervical sympathetic has been found to be altered, and numerous instances of the change have been brought forward by Recklinghausen,<sup>3</sup> Trousseau,<sup>4</sup> Archibald,<sup>5</sup> and others. In eight cases of exophthalmic goitre, referred to by Arnozan,<sup>6</sup> there was degeneration of the cervical sympa-

<sup>1</sup> Edinburgh Med. Journal, February, 1849.

<sup>2</sup> Archiv. für Ophthal., 1857.

<sup>3</sup> Deutsche Klinik, 1863.

<sup>4</sup> Trousseau and Peter, Gaz. Hebdom., 1864.

<sup>5</sup> Med. Times and Gaz., 1865.

<sup>6</sup> Op. cit.

thetic in all; but in four other cases no such lesion was discoverable. In <sup>1</sup>Ebstein's case, as well as those of <sup>2</sup>Reith and Knight,<sup>3</sup> the sympathetic was involved alone, and more often on both sides. Notwithstanding this explanation (the sympathetic origin), others contend that it is a disease of the brain; and still another theory is accepted by those who consider it a cardiac disease *per se*. The nervous origin seems to me to be that which is most acceptable. Not only does the use of galvanic treatment, which cures the disease, suggest the neurotic character of the affection, but the hysterical phenomena mentioned by Basedow, and noticed frequently by others, are certainly significant.

We may, I think, consider the disease to be dependent upon an affection of both the sympathetic and spinal accessory nerves. The condition of the vessels of the thyroid gland and those of the orbit, the flushing of the face, and general disturbance of digestion, are probably due to the altered function of the first-mentioned nerve, and the heart excitement is a consequence of deficient innervation of the accessories.

**Diagnosis.**—There need be no mistake made in the diagnosis of this affection from simple goitre, and after this is accomplished there is nothing else suggested. An inspection of the enlarged thyroid, and the protruding eyeballs, and the detection of the vascular excitement, are sufficient to enable us to say that the case is one of exophthalmic goitre.

**Prognosis.**—A cure is recorded by Cheadle,<sup>4</sup> another by Mackenzie, who also reported a death. Bartholow<sup>4</sup> has cured three patients; Dr. J. P. Thomas,<sup>5</sup> of Kentucky details a very interesting case which ended fatally in five years. Very little can be said in regard to the character of the disease, but it has been cured in certain instances in a year or two. It may last for several years, however, and is essentially a chronic affection. Trousseau, Charcot, and Corlieu<sup>6</sup> report cures, in which pregnancy, uterine hemorrhage, or some such complications occurred during the disease, influencing its disappearance. Of course, the existence of organic cardiac disease gives the affection a very serious character.

**Treatment.**—Galvanism, it seems, has succeeded admirably, and Bartholow has cured three cases by this agent. <sup>1</sup>Eulenburg treated exophthalmic goitre, as early as 1867, very successfully, and Meyer and Chvostek obtained the most happy results. Eulenburg recommends very mild galvanic currents, and he uses from 6-8 elements. I have used the current from 10-15 Leclanche cells, the water column being employed to regulate the same.

<sup>1</sup> Quoted by Eulenburg.

<sup>2</sup> Medical Times and Gazette, Nov. 11, 1865.

<sup>3</sup> Boston Med. and Surgical Journal, April 19, 1868.

<sup>4</sup> St. George's Hospital Reports, vol. iv., 1869.

<sup>5</sup> Richmond and Louisville Med. Journal, 1877.

<sup>6</sup> Rep. by Jaccoud, vol. i., p. 672, 2d edition.

<sup>7</sup> Cyclopædia of Practical Medicine, vol. xiv., p. 102, Am. trans.

Roth<sup>1</sup> reports a case of exophthalmic goitre, the patient being a woman fifty years of age, her menopause having taken place six years before. She became debilitated, suffered from palpitation and sweating at night, and afterwards there was gradual enlargement of the thyroid gland and protrusion of the eyeballs. The pulse was 120, and the temperature normal. It was impossible for her to close her eyelids. The exophthalmos was greater on the left side, and the thyroid was more enlarged on the opposite side.

Galvanism was used, the positive pole being placed on the upper part of the sternum and the negative on the superior cervical ganglion. On the right side ten cells produced no sensation, but on the left, six were sufficient to produce burning. The current was also passed through the back. The night-sweats and palpitation diminished, and she grew stronger. At the end of a month she had gained two pounds in weight, but the reduction in size only occurred in the left exophthalmos and left portion of the thyroid.

Chalybeate preparations, digitalis, ergot, and cod-liver oil are all excellent remedies. Since the appearance of the first edition of this book I have cured one case by ergot, and greatly helped another by the continued administration of the Syrup of hydroiodic acid in doses  $\text{ʒi.}\frac{3}{4}$  ss thrice daily. If galvanism be used, we should bring the sympathetic nerve under its influence by placing one pole (the positive) at the angle of the lower jaw, and apply the negative over the epigastrium or the thyroid.

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<sup>1</sup> Wien. Med. Presse, 1875, No. 30.



## CHAPTER XVI.

## DISEASES OF THE PERIPHERAL NERVES.

## NEURALGIA.

**Synonyms.**—(See special varieties.)

**Definition.**—Neuralgia may be defined as “a disease of the nervous system, manifesting itself by pains which in the majority of cases are unilateral, and which appear to follow accurately the course of particular nerves, and ramify sometimes into a few, sometimes into all, the terminal branches of those nerves.”<sup>1</sup>

Neuralgia is essentially the result of lowered vitality, and is never a consequence of any sthenic condition. This is proved by the circumstances under which it occurs; it taking its origin from general debility, rheumatism, syphilis, or malaria, or some other disease which produces a cachexia. Anstie very justly considers that it is the first expression of a condition which later on becomes paralysis—one being a partial disturbance, or cutting off of the nervous supply; and the other a complete interruption of the nervous force; and it is a familiar fact that neuralgia very often precedes loss of power in parts supplied by an affected nerve.

Neuralgia is, then, a disease in which pain is the prominent symptom, and with which circulatory, trophic, and motorial disturbances may be connected.

**Pain.**—Neuralgic pain is quite distinct from that of any other disease. It is not at all like that of neuritis, which is constant and aggravated by pressure, but it is paroxysmal, and is characterized by a stage of increasing intensity and rapid recurrence, and by a second stage of “wearing out” or subsidence. It appears suddenly, disappears, and returns, being broken by a period of rest. These breaks or intervals of remission become shorter as the attack increases in severity, until the pain *seems* almost continuous. When the climax is reached, the intervals grow in length, and the pain diminishes in severity, and finally subsides. Repeated neuralgic attacks leave the nerve in a hyperæsthetic condition, so that at particular points it is tender and sensitive to pressure.

These foci of exalted sensation have been called by Valleix<sup>2</sup> “*les points douloureux*,” and correspond to the points of emergence of the nerve from its foramen, or at a point when it passes from a deep to a superficial course. The terminal ends of nerves are much more often the seat of this tenderness than any other part. The external ramifications of the supra-orbital branch of the fifth or the small filaments of other nerves—the

<sup>1</sup> Anstie, *Neuralgia*, etc., p. 14.

<sup>2</sup> *Traité des Névralgies*, Paris, 1841.

ulnar and radial for instance—are not rarely painful to pressure. These painful points are met with frequently in cases of facial neuralgia. A gentleman who consulted me some time ago presented this indication of facial neuralgia, there being several hyperæsthetic spots in the roof of his mouth, and his gums on one side were exquisitely tender.

*Circulatory disturbances*, of a quite marked character, are pronounced features of the neuralgic attack. The pulse at first is irritable, small and quite rapid. A species of fluttering palpitation is also present, and the surface is pale and cool. In the later stages of the attack, after the pain has grown decided, the face becomes flushed; the pulse soft, full, and quite bounding; and the eyes may be suffused and bloodshot, should the attack be one of facial neuralgia.

During this stage, and after the subsidence of the pain, the patient may sweat profusely.

*Trophic Disturbances*.—These may be connected with the acute paroxysms, or may result from repeated attacks. Among the former may be pemphigus, and herpetic and bullous eruptions; and among the latter, loss of teeth or hair, or alteration in the coloring matter of the hair, atrophy of muscular tissue, and various cutaneous changes. Charcot and Weir Mitchell, as well as various writers upon dermatology, have called attention to the connection of aggravated neuralgic pain, with various cutaneous diseases. The most striking of these neurotic skin diseases is herpes zoster, in which eruptions of a vesicular character, a cluster of patches are found here and there along the course of the affected nerve. The pain precedes the appearance of the eruption, and may continue during its existence, and for some time after, or there may remain a pruritus, limited to the parts which have been the seat of eruption. The neurotic character of this complication may be proved by its very rapid disappearance after galvanization of the affected nerves, or administration of large doses of quinine.<sup>1</sup> The other trophic alterations, which are secondary, will be considered at a later period.

*Motility*.—Connected with some forms of neuralgia are certain conditions of spasm. In form of facial neuralgia which has been known as *tic epileptiform* or *tic douloureux*, tonic spasm of the eyelid or of the masseter muscles is present as a decided symptom. Convulsive movements of the legs, due to spasms of the flexors, have also been observed in sciatica by Anstie; but in cases in which I have noticed this symptom, it seemed rather a result of excessive pain, and an effort upon the part of the patient to relax the pressure upon the affected nerve. Local spasms are quite common; and the muscles of the face, of the trunk or limbs, and the vomiting of sick headache, are varieties of spasmodic action which may be cited as examples of this kind. In a case lately under treatment, I have been reminded of a condition which I have several times observed—a species of heart pain resembling that of angina pec-

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<sup>1</sup> A form of skin disease lately denominated *pompholyx* by Dr. A. R. Robinson, of New York, is an example of a neurosis of this kind.

toris, and connected with facial neuralgia. With this pain there would be spasmodic contraction of the muscles of the thorax. Mitchell<sup>1</sup> "has encountered from time to time certain forms of neuralgia, accompanied by muscular spasms and extravasations of blood in the affected part. He relates three cases, all occurring in females, and explains the circumscribed hemorrhages by nutritive changes in the walls of the vessels, occasioned by conditions of the nervous system analogous to atrophic changes in the skin and nails in nervous diseases."

Valleix has divided the neuralgias into the *superficial* and the *visceral*, and classifies them as follows:—

#### A. *Superficial.*

1. Neuralgia of the fifth nerve (trifacial or trigeminal neuralgia)
2. Cervico-occipital.
3. Cervico-brachial.
4. Intercostal.
5. Lumbo-abdominal.
6. Crural.
7. Sciatica.

#### B. *Visceral.*

1. Uterine or ovarian neuralgia.
2. Neuralgia of the urethra.
3. " " bladder.
4. " " rectum.
5. " " testis.
6. Hepatic neuralgia.
7. Neuralgia of the heart.
8. " " stomach.
9. Laryngeal and pharyngeal neuralgia.

Among the first group the most important is neuralgia of the fifth nerve, which may also exist with a motor complication, as *tic epileptiform*, or with gastric complications, as *migraine* or "sick headache."

#### FACIAL NEURALGIA.

**Synonyms.**—Face-ache; Fothergill's face-ache; Prosopalgia; Trigeminal neuralgia; Tic douloureux; Migraine; Sick headache.

The supra-orbital branch may be alone affected, and the pain confined to the brow and top of the head, or it may be quite generally diffused over the face and head, the three branches being involved. The first division of the nerves is, however, the most common seat of neuralgia; but it is not unusual for an attack to begin above, and finally extend to all of the divisions of the nerve on one side.

*Migraine*, or "sick headache," presents the following features: The attack may be preceded by some chilliness, pallor, and uneasiness, and is

<sup>1</sup> American Journ. of Med. Sci. lviii. 16.



ushered in by a twinge of pain, which begins just above the eye on one side, and radiates over the head. The pain is often erroneously referred by the patient to both sides of the head, when, in reality, but one-half is affected. Deep-seated orbital pain, photophobia, hemiopia and nausea, with an irritable, thready pulse, and increase of pain, immediately usher in the attack, which rapidly increases in severity; the pulse after a while losing its asthenic character, and becoming full and bounding. The patient's face becomes flushed, and his skin red and sweaty, and in rare cases the sweating is confined to one side of the face. The paroxysms of pain, which at first were separated by intervals of relief, next become almost continuous, but after a time, during which the patient may feel like vomiting, they become less severe, and finally, after his stomach has been emptied, may disappear altogether. The features of an attack of this kind are too familiar to need elaboration. The following case will serve as an illustration:—

Mrs. G. is a delicate, hysterical woman, who devotes most of her time to duties of society. Her domestic affairs are worrying, and the constant excitement of entertaining, late hours, and the management of several unruly children, have so worn upon her that now, at the end of the winter, she is anæmic, "run down," and suffers from want of appetite, insomnia, and general debility. About twice a week, at irregular times, she suffers in the beginning from light pains, radiating from the right eye, and over the head, which become quite severe, and increase during the next hour or two. She usually becomes cold, and bundles herself up in shawls and wraps. Her eyelids feel heavy, and the "skin covering" her "face feels as if it were drawn tightly." She is nervous and irritable, and cannot bear the presence of her children, and is sometimes so depressed that she bursts into tears. She has a vague dread of some trouble, the character of which she does not know. The pain increases in severity, and becomes almost unbearable. Her eyes are hot, and "it seems as if a peg was being driven in from behind." Her face becomes very hot, and her temporal vessels throb. The slightest step she may take in walking so jars her head that it gives rise to intense pain. She "feels as if" her "head would split open." She cannot look out of the window, but lies upon her bed, and buries her face in the pillows. Nothing seems to relieve her. She may lie so for hours, panting for breath, and pressing her aching head. After a variable time, sometimes two hours, sometimes a day, the pain is diminished somewhat, and she becomes nauseated; not because food lies undigested, for she has taken none for some time, but the vomiting is of a purely cerebral character. She attempts to vomit, but cannot bring up anything. The effort at retching jars her body, and increases the pain. After this state of affairs has lasted for some little time, she becomes exhausted, and falls back upon the bed, sweating profusely. The pain grows very much less severe, is dull and throbbing, and finally she sinks into a deep sleep, from which she awakens somewhat relieved.

The variations in pain and circumstances which give rise to the disease have led different observers to apply such names as "rheumatic," "hysterical," "sympathetic," "organic," "syphilitic," and "clavus." These terms have little value, and it seems that a nomenclature based upon the

anatomical situation of the neuralgia is all that is needed, and it certainly would do away with much confusion. Facial neuralgia, unless it be due to temporary exciting causes which may be readily removed, is rather an obstinate affection. It may take a periodic character, especially if it be connected with malaria; or it may be more intense at night, should it be of syphilitic origin. The true attack rarely lasts beyond a few hours, but attacks (especially of *tic-douloureux*) may be so frequent as to become almost continuous. The tendency is, I think, for the disease to become firmly rooted, and to increase in severity. If there be a rheumatic, malarial, or anæmic form, there is no reason why the disease should not subside when these morbid conditions are removed. As to *clavus*, in which the pain is compared to that which would probably follow the driving of nails through the skull, it may be said that this is an hysterical condition, and the patients' descriptions are based upon the workings of a disordered imagination.

There are very few cases of facial neuralgia in which all the branches may not be involved at some time or other. If the neuralgia be confined more particularly to the first and second branches of the fifth, the temples and forehead, upper eyelid, root of the nose, and the orbits will be the points at which the pain will be the most severe. Toothache, above and below, will indicate involvement of the middle and lower branches, and if the lingualis be affected, which it quite rarely is, the tongue will be the seat of the violent pain. The painful points are to be found principally over the supra-orbital notch, the infra-orbital foramen, the "malar point," or in the roof of the mouth, over the mental foramen, and in front of the ear. During the attack it is not uncommon to find hypersecretion of saliva, that fluid passing from the angle of the mouth in great quantity, and when the supra-orbital and infra-orbital branches are involved there may be a corresponding profuse lachrymation.<sup>1</sup> Erb<sup>2</sup> has called attention to the occasional increase of secretion from the nasal mucous membrane. This has been referred by Vulpian to irritation of one of the sphenopalatine ganglia. The patient is nearly always excited and irritable, and if the paroxysms be of frequent occurrence he suffers from insomnia, and is entirely unfitted for his daily occupations. It must not be supposed that the vomiting of migraine has any direct connection with the condition of digestion. The attacks are, however, aggravated by the presence of undigested food in the stomach.

The deep neuralgias of this nerve are very obstinate, and often beyond the reach of any treatment. This is notably the case when the superior maxillary or its orbital branches are affected. The ocular symptoms are then of the most formidable description, and life to the patient is a burden indeed.

The following is one of the most inveterate cases of neuralgia of this kind I have ever observed. The patient's trouble began in 1863, while

<sup>1</sup> Sometimes there is spasmodic closure of the orifice of the lachrymal duct.

<sup>2</sup> Ziemssen's *Cyclopædia*, vol. ii.

at school, and then affected the superior maxillary and infra-orbital branches of the fifth nerve. His sufferings were intense, and after trying almost all forms of treatment, and consulting medical men in Europe and in this country, he consented to subject himself to an operation for exsection. The history he brings, which was taken by the house surgeon, Dr. Peale, of Chicago, details the surgical procedures undertaken.

"Patient has for a long time suffered from neuralgia of supra- and infra-orbital nerves, and the superior trochlear nerve. Prior to this he had a closure of the lachrymal ducts of both sides. He had been in Central America, where he was exposed to severe forms of malaria. About two years ago, Dr. Strawbridge, of Philadelphia, cut off the supra-orbital nerves at their point of exit from the supra-orbital foramen. In either eye there is loss of accommodation, and a high degree of hypermetropia. Prof. Holmes, of this city, after an ophthalmoscopic examination, told him that the veins of the retina were diminished in size.

He still suffers intensely with the infra-orbital nerves, and comes in desiring to have them excised. He receives  $3\frac{1}{2}$  grs. morphia, hypodermically, each day.

*Dec. 18, 1876.* An incision made downward from the location of each infra-orbital foramen to the length of one inch through the tissues of the cheek, the nerves raised on a blunt hook, stretched well out, and chipped off at their point of exit. Ether used as the anæsthetic, collodion and silk sutures to approximate the edges of the incision.

*19th.* Patient suffering from intense pain referred to outer edge of right lower eyelid.

*23d.* Considerable cellular inflammation of right side of neck and face.

*26th.* Considerable discharge of pus from incision on right side of face; swelling very much diminished.

*29th.* Discharge of pus from both incisions has now about ceased; considerable cellular inflammation of right side of face in parotid region. He claims he has still the neuralgic pain, but deeper in the infra-orbital region.

*31st.* Considerable swelling and a great deal of tenderness on either side of the neck below the jaw. Patient cannot move the jaw.

*Jan. 5, 1877.* Face continues swollen, and very painful; thinks he still has the old neuralgic pain on right side. Quantity of opiates in twenty-four hours considerably diminished.

*29th.* Patient again placed under the influence of ether. An incision made on the right side in the site of the old one, and the nerve raised on a blunt hook and divided. Following the operation the pain became severe, and the hemorrhage excessive. For a couple of hours all sorts of efforts were made to stop it, and finally we were obliged to resort to ol. terebinth. and ferri persulph. These, with compresses bound on as best we could, checked it so that it only oozed. A large quantity of anodyne was required to allay pain.

*30th.* There has been no further hemorrhage. *Morph. pro re nata.*

*Feb. 2.* All dressing removed without hemorrhage; wound left open and suppurating; dressed with carbolic acid; pain controlled with morph.



4th. Complains of pain in right temple. P. M. Severe headache; wound dressed twice a day.

11th. Patient had been doing well until yesterday. There was a hemorrhage from the wound in the morning, controlled by syringing with cold water. Last night another very severe hemorrhage; used dry ferri persulph. Has had three hypodermic injections of  $\frac{1}{8}$  gr. morph. each, daily. Ordered iodoform to be sprinkled in wound.

March 27. At 3 P. M. patient was etherized, and Prof. Bogue proceeded to resect the orbital branch of the superior maxillary nerve. A circular flap begun in the old cicatrix on the right side, and curving backwards, laid bare the malar bone. An opening was then made through its quadrilateral surface with a trephine into the antrum; the floor of the orbit was then gouged away and the nerve hooked up and ruptured. There was, following this, hemorrhage. A plug of sponge was then stuffed into the antrum and left. In the evening there was a severe hemorrhage from the nostrils and mouth; the nostrils were plugged. Later in the evening the sponge and plug were removed; the antrum washed out; there was a brisk hemorrhage. Monsel's styptic was freely injected; finally the antrum was again plugged with sponge soaked in the same solution. The eyeball was noticed to project considerably more than its fellow, but the sight was not much impaired. Patient has had, till the present time (10 A. M.), morph. gr. iij, by hypodermic injection. This morning complains of great pain in the eye and upper jaw. Plugs not removed. Ordered whiskey and morph. to allay pain. P. M. Pulse, 76; temp. 103°.

30th, A. M. Pulse, 72; temp. 100°.

Yesterday evening the sponge plugs removed from the wound; no hemorrhage occurred; they were not replaced; water-dressing continued through the night. This morning the wound is suppurating slightly; face not swollen quite so badly. Patient has had one grain morph. by hypodermic injection every 4 hours for the past 48 hours. Water-dressing continued. Patient still complains of great pain in the right eye; swelling is considerable; eye closed, with conjunctiva protruding from between the lids. A pledget of lint saturated with alcohol was laid in wound, and water-dressing continued.

April 1. Is feeling better; wound is suppurating considerably; is not swollen so badly; plugged with lint saturated with alcohol, and the cold compresses continued.

3d. The surface of the wound is covered with healthy granulations. The eye very much improved; can open it; can distinguish objects at some distance.

4th. The patient's condition rapidly improved.

6th. Cavity granulating finely; appetite good; everything appears favorable at this time."

The patient came to New York and consulted me October 17, 1877. In spite of all the surgical operations the pain is as severe as it ever was, the focus of intensity being evidently the orbital branch. The eye is without sight, but no retinal changes can be discovered, except paleness at the fundus. The conjunctiva is injected, and the eye is suffused. I gave him two hypodermic injections of morphia, of one grain each, within an hour, but none of the physiological effects followed, and the pain re-

mained unabated. Nothing remains to be done but deep section of the nerve.

A formidable neuralgia is that connected with spasm of the facial muscles, which has received the name of *tic douloureux* or *tic epileptiform*. The former term is that applied by Benedikt, and has been generally accepted by most writers to express the violent and sudden twinges of pain which are accompanied by very forcible spasms of the facial muscles. These spasms may be of varying degrees of severity. The eye may be tightly closed during the paroxysm, or the face violently drawn to one side. The attacks are generally supposed to be confined to those individuals in whom there is a neurotic predisposition; and Erb, Eulenburg, and others consider *tic douloureux* to be a disease of central origin, which seems very probable for some reasons, but not so much so when we take into account the fact that in some cases the disease may appear and disappear, there being occasionally a long period of quiescence, and then a relapse. Anstie considers that the spasm is not *directly connected* with the pain, but is rather inclined to look upon it as a coincidence, or as a result of the epileptic tendency, the pain and epileptiform spasm being separate expressions.

A very interesting case, to which I have already casually alluded, was sent me by my friend Dr. Sayre, of New York.

Mr. K. had for ten or twelve years suffered from neuralgia of the fifth nerve of the right side. His habits had been very good, and there was no history of syphilis, nor any evidence that it had existed. About ten years ago, after exposure, he first noticed the commencement of his trouble, and at this time there was no facial spasm or very decided pain; his attacks, however, which, during the first two or three years, occurred at intervals of two or three months, became much more frequent, and, within three years, have become almost continuous, so that there is rarely an interval of five or ten minutes between each paroxysm. Sleep is utterly impossible, and he has been obliged to resort to an immense quantity of stimulants for the purpose of procuring rest.

He tells me that very often he drinks a pint of whiskey before retiring. During his visit he had several attacks of tic, during which his face was drawn up and agitated by clonic spasm of the muscles of the right side; these attacks lasted one or two minutes, during which his face became flushed, his eyes injected, and from the corner of his mouth trickled a quantity of saliva; the gum was very tender, and painful points before alluded to were found to be very sensitive. Numerous painful points were also found upon the scalp, over the supra-orbital notch, and at different points over the temporal bone. Before I saw him he had been under several varieties of treatment, but none afforded him the least relief.

#### CERVICO-OCCIPITAL NEURALGIA.

When the posterior branches of the upper cervical nerves are the seat of neuralgia, the patient will complain of pains beneath the occiput, behind the ear, and sometimes at the under part of the lower jaw. The pain at the base of the occiput is most severe; but when the neuralgia in-

volves the anterior nerve branches, and pain appears behind the ear and over the lower part of the face, this affection may be mistaken for neuralgia of the fifth pair. The pain is often insupportable, and is of a paroxysmal character. It is, on the other hand, of a localized form, and so constant in some cases that the medical man may be led to suspect inflammatory conditions of other parts. During the active pain the patient may be unable to turn his head or open his mouth, and any muscular movement is attended with distress. The skin may be either hyperæsthetic or anæsthetic, but more often the former, and I have had patients who were unable to bear even the pressure of a collar or other neck gear. The skin feels to the patient as if it were tightly drawn over the tissues beneath, and it sometimes may be red and appear swollen. The hyperæsthesia, when it involves the scalp, is so distressing that the patient is unable to place his head upon the pillow, or wear a hat unless it is much too large for him; and heat seems to increase the discomfort to a marked degree. The post-cervical muscles may be the seat of cramps, during which the patient's head is drawn backwards or laterally downwards. Painful points may be found in two or three situations, but most frequently where the great occipital nerve emerges. The spinous processes of the upper cervical vertebræ are often the seats of painful spots, and it is not rare to find that distress is caused by pressure at different places over the occipital bone.

#### CERVICO-BRACHIAL NEURALGIA.

A form of attack manifesting itself in severe pains, which shoot down the arms, hands, and back of the neck. Exquisite cutaneous hyperæsthesia is by no means a rare accompaniment, the skin being so tender to pressure that the slightest touch of the clothing will produce intense suffering. The distribution of pain corresponds to the parts supplied by the lower cervical nerves or regions which are innervated by sensory branches of the brachial plexus.

Erb<sup>1</sup> has given a diagram which demonstrates the districts of pain, and their source of supply, which may be made use of in tracing the course of the affected nerves. (See page 534.)

My attention has been directed by Dr. Burral to a condition of neuralgia which is often mistaken for the so-called muscular rheumatism, and is probably due to an involvement of the circumflex as well as the posterior thoracic. The pain is not nearly so acute as that of some of the other neuralgias; for example, the facial variety. It is dull and terebrating, and resembles the agonizing though temporary pain which follows a blow upon the popularly called "funny-bone," or ulnar nerve, in its exposed position at the internal condyle. The pain travels down into the hand, and may be attended by a spasm of the muscles. There are points of tenderness which are extremely numerous. Pressure made over the supraclavicular space, just below the lower angle of the scapula, at the

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<sup>1</sup> Ziemssen's Cyclopædia, vol. xi. p. 146.



exposed portion of the ulnar nerve at the elbow, and at the points of emergence of the superficial nerves of the arm and forearm as they pierce through the fascia, gives rise to pain. Occasionally there are tender spots over the cervical vertebræ. The skin of the arm is often cold, and areas of capillary emptiness are to be observed either during an accession of pain or between the attacks. In rare instances it is not unusual for trophic alterations to be manifested. In a patient under observation the right hand is reduced in size, the skin is dry, puckered and livid; the lines of flexure of the fingers and hand are red, and much deeper than upon the other side of the body; and the nails are crenated and irregular. Erb alludes to an excessive sweating of the fingers. This form of neuralgia is decidedly inveterate, and when well established is attended by nocturnal exacerbations. The use of the affected hand is sure to aggravate or precipitate an attack, and changes of temperature act usually in the same manner.

A gentleman sent to me by Dr. Ives, of New York, had suffered intensely for a number of years, and his pain had become almost constant. When he neglected to cover his arm with cotton batting, but permitted his coat sleeve to come in contact with the skin, he would be in utter misery, so that he was obliged to cover it with some soft substance. He was very cautious in selecting a position at night, as the arm, if unsupported, dragged the muscles of the shoulder sufficiently to produce a paroxysm.

#### INTERCOSTAL NEURALGIA, OR PLEURODYNIA.

This is often mistaken for pleuritis. It is characterized by a pain which encircles the body, and may be referred by the patient to the region bounded by the crest of the ilium below, and the thorax above; but it more commonly affects the lower intercostal nerves. The pain is always one-sided, and is dull and continued, but may sometimes be sharp and paroxysmal, radiating from the spine anteriorly. The skin is hyperæsthetic, and this is particularly the case if the neuralgia be attended by herpetic patches. The painful points are chiefly over the inter-vertebral foramen, and where the nerve pierces the muscles anteriorly. The rectus muscles contain painful spots at the points where the lower intercostal nerves pierce the investing sheaths. The patient during the paroxysm inclines his body to the affected side, as it were to relax the muscular strain; he perspires freely, and his face wears a scared and anxious expression, suggestive of great suffering. His breathing is "catching" and shallow, and attended by the least possible movement of the thoracic walls or diaphragm.

#### SCIATICA.

Sciatica is perhaps, next to facial neuralgia, one of the most troublesome and familiar neuralgias. It rarely begins suddenly, but has a gradual onset, attended by a variety of disagreeable and annoying symptoms. Cutaneous hyperæsthesia, slight fatigue after walking, and "sore-

ness," a sensation of dragging or of heaviness of the leg and foot, and a number of minor symptoms of a vague character precede the actual pain. This is exceedingly severe, and may exist in a dull form, and during its continuance there may be paroxysms consisting of twinges or "darts" shooting down the back of the leg. Should the patient, while sitting, place his thigh so that the nerve shall be pressed against the edge of the chair, the paroxysm may be precipitated. Anstie has divided sciatica into three varieties, one of which occurs during comparatively early life, and is connected with hysteria. It is dependent generally upon over-fatigue, and affects anæmic people. It is the form which attends irregular menstruation, and the pain is quite severe. In this variety I have rarely found any painful points.

Before the fourteenth year neuralgia of the sciatic variety is very uncommon. In 124 cases collected by Valleix, none were under seventeen years of age.

Sciatica of the second variety is a disease of adult life, and is a result either of exposure, or some such cause as continued pressure of the nerve through sitting in an uncomfortable position. It is not rare among business men, or clerks who sit upon high wooden chairs or stools, and who generally do not support their legs by placing the feet upon the floor or the rounds of the chair. Anstie connected this "middle-aged sciatica" with premature decline, and states that the patients have rigid arteries, gray hair, and the *arcus senilis*; but I do not consider that these indications of decay have any very decided bearing upon the sciatica, especially in the form last mentioned. It strikes me rather that the causes which produce the disease, with the exception of dissipation and perhaps syphilis, gout, or like affections, would be local. Some of the most intractable cases of sciatica I have ever seen were persons who were apparently in good general health. The presence of "painful points" is highly characteristic of this form. Foci of tender nerves may be found corresponding with the emergence of the sciatic nerves from the pelvis; and also at various points corresponding to the cutaneous distribution of the posterior branches, as well as just below the crest of the ilium. Points of tenderness may be also found at various situations in the course of the nerve at the back of the thigh; sometimes in the popliteal space, or at the head of the fibula, and in the depression below the external and internal malleoli. Atrophy of the muscles of the thigh is not a rare consequence of the neuralgia in old cases, and is sometimes preceded by paresis. Tactile sensibility is diminished, and areas of anæsthesia or blanching of the skin are occasional results of a continued siege. The paresis of sciatica is of gradual appearance, and the patient may at first slightly drag his leg or limb. In some of the old cases the least movement of the limb is attended by pain, which is referred by the patient to the point where the sciatic nerve leaves the pelvis. Such atrophy may follow inactivity.

A curious feature of the disease in some cases is the appearance of pain in different parts of the limb. In the case of a gentleman who came to me for advice, I found that there were two districts of pain: one

of which included the upper part of the sciatic, the pain never passing below the middle third of the right thigh; the other situated at the outer side of the leg of the same side.

#### CRURAL NEURALGIA.

When the pain is confined to the anterior and lateral parts of the thigh, it is properly included in the cases called by this name, but the region supplied by the crural and its branches, viz., the inner surface of the thigh and its anterior aspect, as well as the inner part of the leg and foot, is more often the seat of pain in the lower extremity than any other part except that innervated by the great sciatic. This pain is paroxysmal, very severe, and, like that of the cervico-brachial variety, most intense at night. The inner part of the leg and foot are most commonly implicated, and there is a subacute variety of pain which exists between the paroxysms. Walking and muscular movements of any kind are painful, and the patient may find it necessary to use a crutch, or else is obliged to keep quiet. Foci of tenderness may be detected at the point where the crural nerve is most superficial, in the groin at the inner side of the knee, at the upper and inner edge of the patella, and at various points on the inner side of the foot and leg. Muscular atrophy, which is probably a result of insufficient use of the limb, is sometimes a feature of the disease. When the pain is more severe at the knee-joint, we may find an enlargement of that articulation, and in some respects the condition may resemble arthritic inflammation; but the cutaneous hyperæsthesia is much greater than in the latter affection, while deep pressure does not produce the amount of pain it would in rheumatism. In many respects the pain may resemble that of posterior spinal sclerosis.

#### THE VISCERAL NEURALGIAS.

The visceral neuralgias, especially those found to be connected with the uterus and its appendages, come more properly within the province of the gynecologist than the neurologist; so a complete description would necessitate a consideration of the various pathological uterine states which would be out of place in this book; therefore our description must be exceedingly brief. The importance of these latter forms of neuralgia cannot be over-estimated. They are commonly of reflex origin, and depend very often upon some morbid condition of the uterus and ovaries themselves. As Anstie remarks: "The amount and force of the peripheral influences which are brought to bear upon the central nervous system by the functions of the uterus and ovaries are greater than any that emanate from the diseases and functional disturbances of any other organ in the body." The menstrual period is that with which neuralgia of this kind is, in nine-tenths of these cases, associated. It is essentially connected with irritability of the pelvic organs of the female, either when there is amenorrhœa and dysmenorrhœa, or when the generative apparatus is over-excited by immoderate copulation or masturbation, or during the pregnant state. When there is any mechanical condition of narrowing or occlusion of the cervical canal, prolapsus uteri, intra-uterine growths,



ulcers or reflected irritation, neuralgia is not at all a rare accompaniment. I have found it very often as a symptom of general anæmia, with no appreciable uterine disease whatever.

#### OVARIAN NEURALGIA.

Ovarian neuralgia is symptomatized by excruciating pains radiating from these organs. It is not necessary that there should be derangement of menstruation, though such is generally the case. The pain may sometimes be dull, but is more apt to be quite sharp. It is greatly increased by standing, or by fatigue following protracted use of the lower extremities. Among sewing-machine operators it is especially common, and many of my cases have been of this kind. It is generally connected with constipation or a sluggish condition of the circulation, sometimes leucorrhœa, hysteria, and always with a great deal of weariness and prostration. The suffering may be so intense and protracted as utterly to wear out the patient, and unfit her for any labor. It may be bilateral or unilateral. There are various other forms of neuralgia which depend upon reflected or local causes.

#### URETHRAL NEURALGIA.

This is not infrequently associated with stricture, gonorrhœa, or masturbation. It may be quite obstinate and of a paroxysmal character, and is much worse at night. I have found it very often where there has been a contracted meatus, in which case the pain ran up the penis. Vesical neuralgia, which may be connected with the presence of a stone, or which occurs as a result of long-standing cystitis, is symptomatized by pain at the neck of the bladder, where there may be some tenesmus.

#### RENAL NEURALGIA, ETC.

Renal neuralgia cannot be diagnosed with certainty, and probably the pain is in many cases due to the presence of calculi. *Neuralgia of the testis* is symptomatized by sharp pains of a temporary character; and it is generally due to some distant source of irritation, such as the descent of a renal calculus, or the presence of a vesical calculus. I have seen cases which have followed excessive venery; and Anstie reports a case of epilepsy in which this form of neuralgia was undoubtedly the exciting cause. Self-abuse produced the "testicular neuralgia," which in turn precipitated the fits. With the pain there were vomiting and great prostration. Ascariæ in the rectum may give rise to neuralgia of that gut. The pain is nearly always about the anus or just above the sphincter, and darts upwards. Cold or exposure are given as causes. The breasts are often the seat of a very painful neuralgia, which has been called *mastodynia*. This is, in reality, a form of intercostal neuralgia, in which case the anterior and middle cutaneous branches of the intercostal of one or both sides are affected. It appears at puberty, or may accompany lactation when the nipples are cracked. In both these classes of cases there must be a lowered nervous condition; and, according to Anstie, masturbation

precedes the trouble in the youthful patient, while it is extremely probable that the strain upon the nervous system during pregnancy and lactation is often much greater than the badly-nourished patient can bear. I have met with the affection in perfectly healthy patients, and am convinced that the pain was purely neuralgic, and not dependent upon any inflammatory condition of the nipples. One of these patients was a prostitute, and had assiduously followed her trade, meanwhile losing sleep, and drinking to excess.

**Causes.**—For the sake of conciseness, I may group the causes which are predisposing and exciting under the following several heads:—

1. Hereditary.
2. General diathetic (anemia, rheumatism, alcoholism, gout, syphilis).
3. Psychological (intellectual, emotional).
4. External (cold, pressure).
5. Sexual.
6. Reflex.

*Hereditary Predisposition* plays a most important part in the genesis of neuralgia, so important indeed that it is difficult to find cases of this disease in whom there has not been some family history of previous nervous trouble. Insanity, paralysis, alcoholism, or convulsive disorders may be traced back; and of twenty-two cases collected by Anstie there were but five in which there had been no family neurotic history, and in some of these phthisis was found. This disease, according to Anstie and others, seems to play quite an important part in the causation of neuralgia; and in one minutely detailed history given by him the appearance of tubercular meningitis and other neuro-phthisical diseases followed the engrafting of the pulmonary trouble upon the neurotic stock. Epilepsy enters extensively into the causation of many forms of neuralgia, especially epileptiform tic; and not only may these other neuroses have appeared among the progenitors of the individual, but they actually exist with the neuralgia.

Blandford<sup>1</sup> has called attention to a form of insanity which coexists with neuralgia, the pains subsiding during acute mental disturbance, and reappearing with its subsidence. Migraine is too common an accompaniment of epilepsy to need more than a passing allusion. Chronic alcoholism is associated with a variety of neuralgic headaches and pains in the lower extremities, which are quite intense. Certain general diseases, which produce a cachectic condition, quite often give rise to the disease, not only by actual mechanical disturbance of the nerve-functions by effusion and periosteal disease, but through the condition of mal-nutrition and enfeeblement of the nervous system which originates in malaria, gout, rheumatism, and syphilis. The influence of malaria in the production of neuralgia is markedly seen in the South and South-west, where the

<sup>1</sup> Insanity and its Treatment, p. 95.

most violent attacks of neuralgia yield only to large doses of quinine and arsenic. The neuralgia is generally of the facial variety, but it may take the sciatic or any of the other forms. In many cases it is periodic, or occurs in connection with the chill and other features of the malarial attack. In most of the cases I have seen, it followed generally after a protracted siege of "fever and ague," when there was extreme debility, "bone-ache," and enlarged spleen.

Lumbo-abdominal neuralgia is far from being an uncommon malarial state, and is sometimes very apt to be mistaken for renal colic. Gout and rheumatism are not looked upon by Anstie as diseases which play a very important part in the general causation of neuralgia, from which opinion I am inclined to dissent. Putting entirely out of question the local inflammation of the nerve sheath, which is so often a cause of sciatica and other neuralgias, I am convinced that there are forms of the disease, aggravated by changes in temperature, coexisting with painful joints and extremely acid urine which disappear under alkaline treatment, and are not clearly examples of nerve-sheath inflammation. Gout, inducing very often a condition of general or cerebral anæmia, has been in my experience, a very frequent cause of facial and other neuralgias. The condition of the liver, which occasions cerebral anæmia, melancholia, and over-loaded bowels, may also induce a neuralgia of a functional character. Not only in the tertiary form of syphilis, but, long before this, neuralgia may often be a troublesome symptom. I have had recently under my care an individual who had two years ago a primary sore, and has since had secondary symptoms. A chancroid, recently contracted, assumed a phagedenic character, and there were great debility and severe neuralgia, which succumbed under specific treatment and nourishing diet. Profound anæmia is very often found to be the origin of neuralgia of various kinds. In women who have lost much blood during the menstrual flow, or in others who have become exsanguined from hemorrhoids, neuralgia is not to be looked upon as an unusual complication.

The various constitutional diseases just alluded to may produce various forms of neuralgia, by inflammation of nerve-sheaths, with deposit, or, as in the case of syphilis, gummatous growths; or periostitis may make dangerous pressure upon the nerve-trunk at some point where the latter is unable to withstand it without injury to itself. Syphilis, in rare instances, produces irritation in the nerve-trunks themselves, giving rise to pain. This irritation, however, much more frequently produces motor paralysis than sensory disturbance. Mental overwork, shock, and a continued abnormal play of the emotions are likely to give rise to neuralgia, and for this reason literary men and hysterical women suffer very frequently. The headache of the overworked school child, compelled to overtax its brain, and dependent upon confinement in a hot room, is far too common. Want of amusement, deep grief, and the pursuit of one narrow line of thought, are all influences which lower the integrity of the nervous system, and give rise to this as well as other neuroses. Anstie's practical and judicious reasoning in regard to false religious training, and the dangers



it may bring in the way of forcing the individual to become self-conscious, should suggest to the physician and parent the necessity for avoiding everything in education which promotes brooding, causes the individual to torture himself with doubts and self-accusation, and narrows the mind, thus depriving the nervous system of its *normal* exercise. Constant worry about business and any strain which demands an unusual expenditure of brain-force are causes of this kind. Exposure to cold and damp, particularly if there be wind, is a fruitful exciting cause of neuralgia, and persons who are exposed to draughts in railroad cars and public buildings very often owe their attack to such agencies. Pressure from various growths, cystic, cancerous, and gummatous deposits, not rarely causes distressing and intractable neuralgias; but a syphilitic growth has been known to entirely surround a nerve-trunk without interfering materially with its functions.<sup>1</sup> Neuromata very frequently give rise to neuralgia. Such neuromata sometimes follow amputation or gross nerve-wounds, and the neuralgia is generally relieved by extirpation of the nerve-tumor. Various local troubles of a peripheral or remote nature, produce neuralgia, and among these may be mentioned carious teeth, ascarides, and renal calculi. When carious teeth give rise to neuralgia, it is always very obstinate, and the cause may remain unsuspected for a long time.

Salter has observed cases of cervico-brachial neuralgia from bad teeth; the variety most frequently met with however is facial neuralgia. This cause is ordinarily supposed to account very frequently for the head neuralgias, and many sound teeth are sacrificed by the individual, while there may be neuralgia of the two lower branches of the fifth from other causes. Over-use of the eyes, and consequent fatigue of the muscles of accommodation, are supposed by some to have much to do with its production. Renal or urethral calculi, gonorrhœa, masturbation, and excessive venery, are all reflex causes of importance, and play a part in the production of lumbo-abdominal and other neuralgias. Uterine disease and overloaded bowels, or a fibrous tumor in the rectum, may by pressure often produce sciatica of a very obstinate variety, and aneurism more rarely makes pressure which gives rise to neuralgia. Digestive derangement and prolonged lactation may be mentioned as additional conditions which favor the production of neuralgia. As to age and sex, it is the opinion of most authors that neuralgia usually originates at the age of puberty, but the disease is most common between the twentieth and fiftieth years. The following table, presented by Erb (Ziemssen, vol. xi.), possesses statistical value:—

|                                | Valleix. | Eulenburg. | Erb. | Total. |
|--------------------------------|----------|------------|------|--------|
| Period of life up to 10 years, | 2        | 6          | —    | 8      |
| “ “ 10 to 20 “                 | 22       | 19         | 14   | 55     |
| “ “ 20 to 30 “                 | 68       | —          | 40   | 108    |
| “ “ 30 to 40 “                 | 67       | 33         | 39   | 139    |
| “ “ 40 to 50 “                 | 64       | 23         | 29   | 116    |
| “ “ 50 to 60 “                 | 47       | 14         | 14   | 75     |
| “ “ 60 to 70 “                 | 21       | 6          | 9    | 36     |
| “ “ 70 to 80 “                 | 5        | —          | 1    | 6      |
|                                | 296      | 101        | 147  | 543    |

<sup>1</sup> Huebner Ziemssen's Cyclopædia, vol. xii.

As to sex, Valleix collected 469 cases, 218 of whom were men; Eulenburg 106, of whom 30 were men; Anstie 100, of whom 33 were men; Erb 146, 84 being men. Of course there are varieties of neuralgia which are confined more to certain ages and sexes. Migraine is more general among women, while sciatica is probably more often a disease of males. Anstie considers facial neuralgia to be a disease of adult life. So far as climatic influences are concerned, neuralgia is predisposed, and very often markedly affected by sudden changes in temperature. Dr. Weir Mitchell<sup>1</sup> has written a very valuable paper upon the subject, which clearly shows the very decided influence of modifications of temperature and humidity. His article is based upon the personal notes of Captain Catlin of the U. S. Army, who suffered from stump neuralgia, and who intelligently and carefully noted the influences of atmospheric changes. Captain Catlin's conclusions were as follows: "Neuralgic intensity does not seem to be proportioned to the amount of rain-fall. At the exterior of a storm disturbance the pain is usually severe, and, indeed, at times I have been so far from the disturbed centre as to just perceptibly feel it. A storm, reinforced by another at an angle of say 90°, producing greater eccentricities in the curves, does not seem to produce a corresponding intensity of duration of the neuralgia." He adds: "I am unable to state at what point within the disturbed area the pain would be strongest. The abruptness of the barometric fall does not seem to have much to do with the causing of pain, nor is the length of attack dependent as it seems on the length of the storm."

**Pathology.**—Neuralgia is always the result of lowered functional activity dependent upon the trophic disturbance of a sensory nerve. This is probably attended by some change in the posterior nerve-roots, which is not necessarily inflammatory. The morbid anatomy of neuralgia has thrown but little light upon the pathology of the disease, so our conclusions must be based upon purely theoretical grounds. Erb, in speaking of the nutritive disturbances, says: "In regard to the ordinary seat of this trophic disturbance, nothing accurate is known; but it is probable that the seat varies, and this much appears certain, that for the most part a definite group of fibres (or their central terminations) as they are combined to form a nerve-trunk or branch, is affected. At what place in the length of the nerve this is present it is difficult to say, and perhaps may be at any length. The peripheric fibrils may be affected at various points and various lengths of their course, or the posterior roots and their prolongation in the spinal cord may be the seat of the neuralgic trophic disturbance; or, lastly, the central fibrils running in the spinal cord or brain may be affected up to the terminal central apparatus. The investigations that have hitherto been made have acquainted us with many important facts, but have furnished no very satisfactory conclusion."

The clinical features of neuralgia enable us to understand many of the phenomena which ordinarily characterize the disease, and we are permitted

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<sup>1</sup> American Journ. of Med. Science, April, 1877, p. 305.

to assume that lowered nutrition from general or local disease, reflected irritations, and mechanical pressure enter into its production. Instead of a normal stimulus being conveyed by a healthy nerve to the centre, the nerve may be functionally impaired for conduction, or the centre so altered in its receptive faculty that the sensation period is grossly exaggerated. The receptive faculty of the peripheral fibrils may be so exaggerated that ordinary stimuli are received and transmitted in a painful form. Why the disease should be paroxysmal we do not know.

Of late much discussion has followed the presentation of a new instrument by Vigoroux for the treatment of neuralgia, and the nerve-current theory has been the subject of earnest inquiry and speculation. In this *percuteur* a small hammer is made to tap the surface of the body over the neuralgic nerve, and, while rapid tapping relieves dull pain, slow tapping is most efficacious in violent neuralgic pains. In the healthy subject any kind of tapping produces pain where none existed before. Granville and Vigoroux, both of whom claim to have invented the instrument simultaneously, hold that neuralgia is the result of an irregular current wave or vibration.

**Morbid Anatomy.**—It is by no means a matter of necessity that a nerve which has been the seat of neuralgia is found to be changed in structure. Accidental atrophy, hyperæmia, and indications of neuritis are sometimes exhibited. Thickening of the nerve and sheath deposits in its neighborhood, or enlarged vessels, tumors, aneurisms, and the like, are occasionally met with. On the other hand, nerves have been removed which have been perfectly healthy. In old cases of neuralgia the posterior nerve-roots are nearly always atrophied.

**Diagnosis.**—We may briefly sketch the character of the symptoms. The pain of neuralgia is paroxysmal or dull, with paroxysmal recurrences; rarely tenderness upon pressure, except at certain situations. Neuralgic pain is rarely constant, while that of neuritis is quite so. The pain of neuralgia follows the course of some nerve, is quite acute, and has a lancinating, terebrating, or shooting character. It is also connected with vasomotor changes in the skin. The existence of a cause must be considered, and the fact whether “hereditary predisposition” is present or not. Facial neuralgia is very rarely mistaken, and should not be when the fact is taken into consideration that the pain is generally referred to one of the branches of the fifth nerve. Pleurodynia is sometimes confounded with pleuritis, but the absence of physical signs should be sufficient to make the diagnosis clear. Lumbo-abdominal neuralgia is very frequently confused with various painful affections of the viscera. Among these may be mentioned renal colic, the pain of nephritis, and intestinal colic. Sciatica, from its unilateral character, is not likely to be mistaken for any other affection. The important indication in diagnosis is to determine the variety of neuralgia, whether syphilitic or malarial, whether due to compression or connected with neuritis, or whether due to enlargement of, and pressure from, any of the abdominal organs.



The following are to be remembered and consulted for guidance in making a diagnosis—

- A. Cause ; history of previous attacks.
- B. Character of pain ; paroxysmal, inconstant.
- C. Aggravation by debility or fatigue.
- D. The presence of "painful points."
- E. Its distribution (following course of nerves).
- F. Rarely aggravated by pressure, except at limited points, which correspond to superficial course of the nerve.
- G. Its general unilateral character.

**Prognosis.**—Neuralgia of all kinds is more curable in early life than in advanced age, and it may be assumed that, when it has lasted for many years, and is severe in character, it will be most intractable ; this is especially the case in the disorder known as *tic epileptiform*, which may be said to be nearly always incurable. In these troublesome cases even removal of the nerve affords but temporary relief. When atrophy of muscles has taken place the chance of cure is very remote, and if the cause be a deep one, such as pressure for instance, nothing can generally be done. There is a bright side of the picture however. Functional neuralgias, or those of the syphilitic variety, readily succumb to proper treatment ; and sometimes general nourishment and the removal of the exciting cause will speedily restore the patient to his normal condition.

Those neuralgias which develop later in life are attended by structural decay, arterial degeneration, and are very hopeless. As to the curability of the varieties of neuralgia, that of the fifth nerve is most persistent, and intercostal neuralgia perhaps least so, whilst sciatica holds a place midway between the two. As an example of a severe and intractable continued neuralgia, connected probably with angina pectoris, I may present the case of

Lucy L. S., sixty-five ; U. S. ; married. *Previous History.*—When a young child she fell, striking her right eye on a chair-post. For several days it was supposed she had lost her sight, but this was found not to be the case. After this she had pain in the left side and shortness of breath, whenever she attempted to run. At twenty-one she had an attack of cerebral hemorrhage, which affected the right side, but there was no aphasia. This was accompanied by anæsthesia, which has never entirely disappeared. About this time there were diplopia and ptosis—the latter symptom being now present. Supposed pulmonary trouble at twenty-four. Married at twenty-five.

"Before birth of my second child, I was subject to dizziness, and neuralgia of the fifth nerve, which was most intense in the morning.

When nearly twenty-eight, and my second child was a few days old, I 'commenced to see dark spots, sometimes like black specks, again like circles with spotted centres.' When this child was three or four weeks old, sharp pain commenced in right side of the head. After sleep the pain would subside, and vision would improve. At intervals of from three to four weeks, or when tired, these blind attacks would return, accompanied either by sharp pain or dizziness in the head. For the next eight years

I was comparatively well, having occasional 'blind turns' when tired. At these times my forehead would feel as if strings were being pulled in opposite directions, and there was much twitching in the right eye. All these years there was some pain about the heart, with palpitation.

At forty-one the change of life commenced, and I suffered several years most intensely.

All these years there was some difficulty around the heart. Palpitation and some pain at intervals.

For the past three years pain has been about equally divided between head and heart; sometimes commencing in one and sometimes in the other. Some six months ago pain seemed to be settling around heart particularly. Would come on with a chill and creeping sensation up the spine, and would begin with a whirling in left side. A palpitation of the heart would come on if excited or tired. Outward applications and medicine taken seemed to drive pain across from left side to right shoulder. Would go into right side of the head; follow down right arm into hand. Also into left arm and hand. Hands have been much drawn up, and streaked with red. When pain was in face it would be spotted red and white on right side only. When severest in side and heart, eyes became set in head; face livid, and blood would settle under nails. After enduring pain, tremble much in limbs."

I saw the patient during the past spring, and found her to be a rather spare, badly-nourished woman, and she presented the following symptoms:—

*Objective.*—The right eye was examined and found to be sightless; the retina was the seat of an old neuritis, with atrophy of the disk. There was slightly developed ptosis of this eye, and some keratitis, corneal opacity, and ulceration, and she was obliged to wear a shade. The right side of the face was slightly anæsthetic and analgæic. Æsthesiometer contact and extremes of temperature were not readily perceived. The same was the case in the skin of the right arm, forearm, and hand, but more decidedly the latter. The hand presented the appearances to be hereafter described (see article upon NEURITIS), and was markedly anæsthetic, and the skin showed evidence of impaired nutrition. The right lower extremity was in much better condition. There was very slight loss of motor power on the right side.

*Subjective.*—She now has attacks of severe facial and cervico-brachial neuralgia which come on every two or three weeks, and has had one within a day or two; there is still some tenderness left in various parts of the face and right upper extremity. The pain seems most intense in the upper branches of the fifth, and has never affected the inferior maxillary to a decided degree. The arm-pain and head-pain are simultaneous in their onset, and are preceded by the ordinary prodromata of an attack of this kind. They are always paroxysmal, and seem to reach a climax and then subside. During the attack the eye is seemingly "forced forwards." After the attack she is entirely free from pain. With the seizure there is cardiac trouble, and respiratory trouble which suggests some impairment of the pneumogastric.

She never has convulsions or vomiting, and there is no deep, localized pain at any point in the superior aspect of the cranium; but all pain

at this point is superficial, and would evidently come under the head of hyperæsthesia.

In this case there is a decided hereditary history of nervous disease.

**Treatment.**—In nine-tenths of the cases of neuralgia the management of the disease should be undertaken with the assumption that the pain is due to lowered functional activity and depressed tone; and while local treatment is not to be forgotten, it is absolutely imperative that the patient should be supported, and that drugs which improve the nutrition of the nervous system should be selected. It is well to minutely inquire into the existence of other disease, and reference to what I have already said about etiology will furnish the reader with such hints as may be necessary. Should menstrual irregularities, gastric derangement, or constitutional diseases be found, it is well, I may say absolutely necessary, that these should be corrected before any local treatment is to be undertaken.

Neuralgic pain is very variable; and although, for my present purpose, I shall make use of two expressions to denote its character, there is much that must necessarily remain unsaid in regard to its variation and peculiarities.

I shall describe the pain of neuralgia as *coarse* and *fine*, two divisions which, though somewhat arbitrary, are useful when we speak of treatment. *Fine* neuralgic pains may be said to be those of a sharp paroxysmal character, leaving behind no points of tenderness, and entirely unconnected with any suspicion of neuritis. *Coarse* neuralgic pains may be said to include the *brusque* pains, which bring local tenderness and soreness, and are aggravated by movement. The former are those which sometimes occur during migraine and functional neuralgia of the lighter kinds; while the *coarse* pains may be often the result of sciatica, in which the movement of the limb in walking or the pressure of the chair is sufficient to give rise to them. In one form of the latter our treatment should be quite negative, and of a character which necessitates the use of counter-irritants, such as blisters and the actual cautery; while the former is best treated by remedies which either increase the blood-supply of the nervous centres and improve their tone, or allay reflex irritability. The treatment of facial neuralgia or migraine should be the following: The use of diffusible stimulants; muriate of ammonia being, perhaps, one of the best. It should be given in large doses quite frequently, beginning with from twenty grains to a drachm, which should be repeated every hour during the attack. Coffee and tea, or their alkaloids, are often serviceable; or we may prescribe guarana, which is a very valuable remedy, in doses of half a drachm to a drachm every hour. I have never witnessed any bad results from the use of this drug, even when quite large doses were taken. The powder is the best preparation. Tr. belladonna given in small repeated doses, does much good if the disease be of a reflex character. The drugs recommended for this variety of neuralgia are quite as numerous as most of them are useless. The alkaloids





SUPERFICIAL POINTS AND CUTANEOUS AREAS OF NERVE DISTRIBUTION.—1, 2, 3, 4. Points for galvanization of fifth nerve. 5. Brachial plexus. 6. Musculo-cutaneous. 7. Median. 8, 9. Ulnar. 11, 12, Crural. 13. Peroneal. 14. Tibial. 15. Occipital. 16. Radial. 17, 18. Sciatic. 19. Popliteal. 30. Peroneal. *ac.* Acromial. *Cr.* Circumflex. *Int. h.* Internal humeral. *Ext. c.* External cutaneous. *Int. c.* Internal cutaneous. *c. p.* Cutaneous palmaris. *p. u.* Palmaris ulnaris. *m.* Median. *Rad.* Radial. *u.* Ulnar. *Mus. Sp.* Musculo-spiral. *Ilio-Hy.* Iliohypogastric. *I. I.* Ilio-inguinal. *Lat. Cut.* Lateral cutaneous. *E. S.* External spermatic. *Lum. I.* Lumbo-inguinal. *Pos. C.* Posterior cutaneous. *ob.* Obturator. *Com. p.* Communicating peroneal. *Int. sa.* Internal saphena. *Sup. p.* Superficial peroneal. *cpm.* Posterior median cutaneous. *Cpp.* Cutaneous plantaris proprius. *Pl.* Plantaris lateralis.

daturine and conia have been used in obstinate cases of *tic epileptiform* with varying degrees of success, but great care should be taken. I have often broken up an attack of ordinary facial neuralgia with a cup of strong hot tea, or even a cup of hot water; and now have a patient who has been in the habit of taking an emetic, which has almost immediately given her relief. *Cannabis indica*, either in the form of the extract or tincture, is of service when guarana fails. Its use should be continued for several months. If the neuralgia be malarial, a fair dose (say twenty grains) of quinine rarely fails to abate the paroxysm. As local applications, various stimulating liniments are used, the best I know being the compound soap-liniment; or a mixture of chloroform, tr. aconite and camphor; an ointment of veratria or of chloral and camphor sometimes affords relief, and I have witnessed the good effects of a tincture made of the berries of the belladonna. The blister or actual cautery may be brought into requisition if painful points are found, and I have been in the habit of using the ether spray just in front of the ear in migraine. In *tic douloureux* I am convinced there is no better remedy than gelseminum given in large doses, beginning with ℥viii to ℥xv of the tincture or fl. extract. My friends Drs. Kinnicutt and Clymer have both mentioned to me the details of cases where by accident the patient had taken toxic doses of this drug. In one of these the disease entirely disappeared after the alarming effects of the remedy had passed away. Croton-chloral, which has lately been recommended for facial neuralgia, I am convinced has been overpraised; I have given it a fair trial, and have rarely found it of any use. If it is employed twice a day in twenty-grain doses, it will do more good than in the small repeated doses. The removal of carious teeth is often followed by speedy disappearance of the disease. Should the face become tender, as it not uncommonly does, the patient should be directed to keep it carefully protected by cotton-batting; and if painful points remain in the roof of the mouth or gums, they may be lightly touched with the hot glass rod or iron. The treatment of cervico-brachial, cervico-occipital, and other neuralgias of the trunk may be managed after very much the same plan. In each particular case of course the treatment varies. If there be a diathetic condition, such as syphilis, mercurial inunctions, baths and specific treatment are to be made use of in conjunction with local applications. The advantage of large doses of quinine in cachectic headaches, as well as in intercostal or lumbo-abdominal neuralgia, especially if there be an herpetic eruption, I have mentioned. In these forms, as well as in

ovarian neuralgia, the use of local cold, such as may be obtained by ice-bags, or the application of blisters, is very efficacious. The actual cautery, employed to make sweeping strokes along the course of the nerve, or down the back on either side of the spinous processes, and in paths which run at right angles to the longitudinal "stripes," may be brought into requisition, and applied twice or thrice weekly. Sciatica sometimes demands most obstinate treatment. The actual cautery, and even nerve-stretching, may be necessary; but in the majority of cases galvanization of the nerve does great good, and should be faithfully tried before anything else is done. In neuralgia of the rectum it will often be found that stretching of the sphincter ani will effect a rapid cure, especially when fissure exists.

Electricity affords very decided relief in this disease; and galvanism, when judiciously employed, rarely fails to modify, if not cure neuralgia. In facial neuralgia it should be applied to the nerve by small sponge-covered electrodes, one pole being placed just behind the condyle of the jaw, and the other held for a few minutes over the supra-orbital and infra-orbital foramina, or over the symphysis of the lower jaw. The current should be the direct (from positive to negative, the negative pole peripheral). The plates of Morgan, and the suggestions of Ziemssen, will enable the reader to comprehend the situation of the points corresponding to the superficial course of the various nerve-trunks, so that they shall be brought most readily under the influence of the current. Faradism of the intercostal nerves, and of regions of distribution of terminal filaments of other nerves in various neuralgias, is of great service, and rarely fails to afford relief in sciatica. I have seen pleurodynia disappear in ten minutes after the use of the faradic current. The following case shows the benefit of electrical treatment.

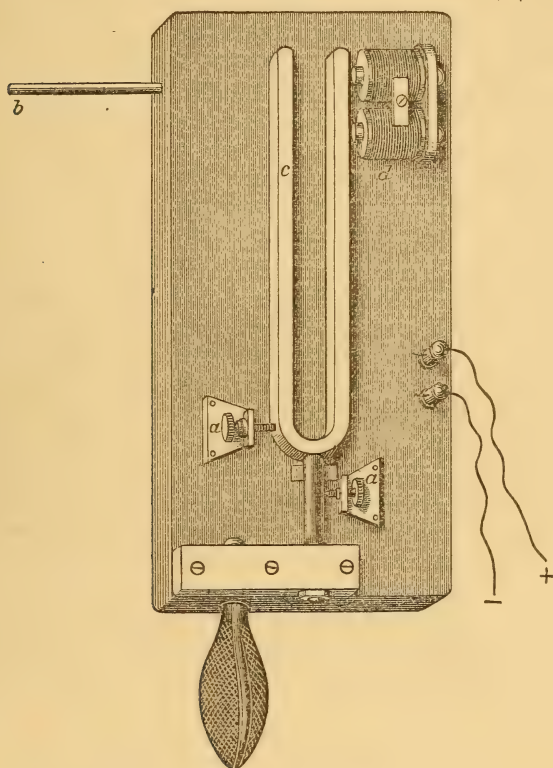
Mr. S. After constant exposure during the war, the patient contracted a low typhoid fever which left him weak and emaciated for a long time. Since 1868 he has had twinges of pain down the back part of the leg, which have left him in a perpetual state of misery, with only occasional intervals of several months when he is absolutely free from pain. In winter his trouble is worse, and any exposure will immediately produce a severe attack of neuralgic pain. Any indiscretion in his diet will also be followed by the sciatica. He had gone through the usual siege of medication, including morphine, hypodermics, and stimulating lotions. He came to me in July, 1871, when I made applications of galvanism to the nerve by the conical sponge-electrode, the sponge being held firmly over the obturator foramen. At the first visit his pain was excessive, but after fifteen minutes' application he left, feeling a sense of relief which he had not known for months. Two months and a half of this treatment were sufficient to dispel the pain, which did not recur. Four months afterwards, he made a visit, when he stated that he had not had any return.

Less than one year ago Granville described an instrument for the



treatment of neuralgia, which effects the mechanical transmission of shocks to a nerve which may be the seat of neuralgic pain. I have not seen Granville's instrument, which consists of a hammer driven by a ratchet wheel, and so regulated that rapid or slow shocks may be made. I have, however, carefully followed out his experiments with an instrument which consists of a tuning-fork, (*c*) vibrated by electricity, and solidly

Fig. 67.



mounted upon a board. The board is provided with an arm, (*b*) which can be applied to the superficial part of the nerve. By means of set screws (*a. a.*) coarse or rapid vibrations may be produced. (*d*) is an electromagnet. In acute pain the slow vibrations are communicated to the nerve trunk, destroying the irregular character of the painful impression, and often affording instant relief. The curious reflex phenomena that result sometimes are indicative of a very profound nervous impression. In my personal experiments I was able to provoke a synchronous vibration in the tensor tympani muscle with subjective throbbing and noises; and an ocular impression manifested in momentary flashes such as are produced by the galvanic current.

In the treatment of neuralgic attacks the hypodermic syringe has played a very important part. I have no doubt that it has been abused, and I have become painfully aware that individuals have thus acquired the habit of opium and morphine self-administration. For the radical cure of certain varieties of neuralgia, the hypodermic syringe has no equal. Dr. T. M. B. Cross, was the first, I believe, to use deep injections of morphine in sciatica. He has recommended that the point of the syringe needle be carried down to the sheath of the nerve, and the contents of the barrel gradually expelled. Strange to say, very few accidents have followed its use, although the wounding of an artery is not an impossibility. Chloroform has been used hypodermically by Bartholow,<sup>1</sup> and with great success, and though I have produced abscesses in this way, I am inclined now to acknowledge its value as a therapeutic measure. Morphine, and atropine, ergotine, and other alkaloids are constantly used, and sometimes afford relief, which is generally temporary, but occasionally permanent. The general treatment, is however, all-important, and iron, strychnine, arsenic cod-liver oil, and phosphorus rank high as valuable remedies. I have spoken of quinine. I may add that when given continuously, either in combination or alone, it cannot fail to do good. Tonga, the new Fiji remedy, has been recently recommended. It is excellent, especially in facial neuralgia, and may be given in doses of from  $\text{m}_\text{x}$ — $\text{m}_\text{xx}$  every two hours until relief is obtained. Phosphorus always does good, except in forms of neuralgia, which are not directly dependent upon depraved nutrition, and are due to cold or attended by inflammatory conditions. Marey<sup>2</sup> has recommended the nitrate of aconitia for facial neuralgia. He has cured cases very rapidly by the administration of a quarter of a milligramme several times a day, increasing the dose until the patient finally took as much as two milligrammes. Gubler also used aconitia in facial neuralgia with much success. Dr. Séguin some time ago called attention to its virtue in neuralgic affections of the fifth nerve. It should be given in doses of  $\frac{1}{100}$ th of a grain, and repeated until the face becomes decidedly numb. The solution used by Séguin is as follows:

R Duquesnel's aconitia gr. one-twelfth.

Alcohol, }  
Glycerine, }  $\bar{a} \bar{a}$                        $\bar{3} \text{ j.}$

Aq. menth. pip. ad  $\bar{3}$  ij. M.

Sig.: One teaspoonful three times a day.

I have used it in the form of saturated tablets, prepared by Caswell, Hazard & Co. of this city.

In cases of headache of the congestive variety it will be found that

<sup>1</sup> Mat. Medica and Therapeutics, p. 321, et seq.

<sup>2</sup> Thèse de Paris, 1880.

tincture of *Cannabis Indica* brought to the physiological point does much good. Thompson's solution is the best preparation.<sup>1</sup> Salt air, with alternations of mountain air, nourishing diet, which should include a large proportion of non-nitrogenous food, attention to the daily habits, the removal of fecal accumulations, and the re-establishment of menstrual regularity are of the greatest importance, and should be accomplished if possible.

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<sup>1</sup> R Phosphori gr. ss.—iss.

Alcohol absolut. q. s. ut. diss.

Ess. menth. pip. q. s.

Glycerinæ ad.  $\frac{3}{4}$  iv.—M.

Sig: A teaspoonful after eating.



## CHAPTER XVII.

## DISEASES OF THE PERIPHERAL NERVES (CONTINUED).

## NEURITIS.

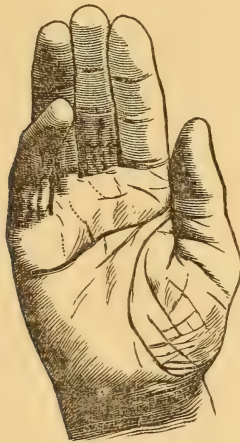
**Symptoms.**—Inflammation of a nerve is expressed chiefly by *soreness* and *tenderness*, and not by darting or paroxysmal pain, which constitutes neuralgia. When confined to the nerve-trunk, various depraved conditions of sensibility, motility, and trophism may follow, which are expressed by cutaneous and muscular changes; and the course of the nerve can usually be marked with great exactness, for pressure produces great pain. The skin may be red or the seat of bullous or pemphigous eruptions. Of course very much depends upon the character and importance of the nerve affected. Some of the nerves of sensibility, such as the fifth, when subject to neuritis, are followed by symptoms different from those which occur when the seventh or one of the mixed nerves is affected. Peripheral inflammation of the external portion of the seventh is often the cause of facial paralysis, and neuritis of the fifth may occasion disorders of sensibility as well as ulceration of the cornea and other trophic phenomena. With neuritis there is not infrequently loss of tactile sensibility and sense of appreciation of temperature, though in the beginning the skin is hyperæsthetic, and the pain is aggravated by contact with cold or hot substances. Erb speaks of acute and chronic neuritis, the former depending upon traumatism, sloughing, or cancer, and beginning with a chill, followed by fever, headache, and sleeplessness. The pain commences in the affected member, and extends, until finally chronic neuritis is progressive, the inflammation spreading, and involving new nerves. This extension may be recognized by the fresh appearance of pain in new localities; by painful points (Valleix's) at new regions, by difference in the form of pain, and by variations attending pressure; the whole limb is affected. This author, as well as Mitchell, considers that it is most intense at night, and that it is augmented by movement. Mitchell has observed intense hysterical excitement, and even delirium. A red line usually marks the course of the affected nerve, and there may be patches of herpes or pemphigus, or the skin may be œdematous. In one case, observed at the Epileptic Hospital, the patient, a negress, presented symptoms of neuritis of the anterior tibial nerve, and the skin of the fore part of the right leg was tense, shiny, and exquisitely sensitive. A marked rigor ushered in its development, and there were subsequently nausea and vomiting. Her pulse was feeble and rapid, and she could not sleep, and entirely lost her appetite. There was no inflammation whatever of the skin or muscular tissue, and the acute pain subsided in a few weeks, but

there remained a condition of great tenderness. Hot and cold applications increased the pain.

Paralysis may follow, and is by no means uncommon. The patient generally recovers in a month or so, and sometimes in a shorter time, but the neural condition never entirely disappears. In the *chronic* form the onset may be gradual or spontaneous, or follow an acute attack. I have sufficiently sketched the symptoms, and will only add that muscular cramps, tremor, or permanent contractures sometimes form very distressing sequelæ, and with these there is paralysis. Anæsthesia or hyperæsthesia is connected with neuritis, the former being of late appearance. Erb calls attention to the comparative immunity of the motor nerves, as paralysis does not follow until after a long train of sensory disturbances, but reflex disturbances are not uncommon. These may consist in remote nerve pain, cramps of distal muscles, or hysterical attacks. The electric excitability in the early stages is exaggerated later, or it is lost, and if there be paralysis there is very marked muscular atrophy as a consequence, and electric contractility disappears altogether. By far the most interesting changes are those of a trophic character. Weir Mitchell has presented a most complete description of these structural alterations. The finger-nails lose their normal character, and become horny and curved, and the skin becomes rough and is sometimes exfoliated.

As additional evidences of this defective nutrition, "hang nails," cracking of the skin and other slight changes from its healthy condition are striking indications. The illustration (Fig. 68) which I produce is

Fig. 68.



Trophic Change of the Skin.

from the photograph of a patient whose hand had been anæsthetic for some years. The skin is hard, the palmar furrows are sharp and exaggerated, and the bases are red or purple, somewhat resembling the same

appearance in the cutaneous flexure of the knee, elbow, or other articulating parts in certain forms of chronic eczema.

**Causes.**—The acute variety is dependent upon injuries of various kinds. I have seen one case which followed a carbuncle situated upon the inner surface of the forearm, and Mitchell reports several cases following gunshot wounds. Flying splinters, fractures, and blows are various traumatic causes, while the extension of cancerous disease or sloughing may produce a neuritis. Cold, rheumatism, and syphilis enter into the etiology of the affection, and Mitchell has produced a neuritis by the local application of ice. In one case of facial spasm, for which I used the ether spray, I was disagreeably surprised to find a remaining neuritis of the portio dura, which lasted for some time.

Beau has directed attention to forms of neuritis of the intercostal nerves which undoubtedly arose from pleurisy and pleuro-pneumonia. Typhoid fever, diphtheria, and other diseases of a febrile nature are not infrequently attended by neuritis, and in one case of typhus, reported by Bernhardt, a neuritis involved the musculo-spinal nerve.

**Morbid Anatomy and Pathology.**—Inflammation of a nerve-trunk produces very decided changes in its appearance. It becomes swollen, is of a pinkish hue, and there is often an exudation which is found between the fasciculi; this may be also of a reddish color. The microscopical appearance of the nerve is still more characteristic. The nerve-fibres undergo marked changes; the axis, cylinder, and the medullary contents are disintegrated; the neurilemma may be distended by serous exudation, and the blood vessels are enlarged and in places ruptured, so that blood-elements may be found scattered in different regions. In later stages there may be atrophy or fatty degeneration. In chronic neuritis these appearances of advanced degenerative changes are found to consist in proliferation of connective tissues, and this takes place as an interstitial formation. Degeneration of the minute nerve-elements, deposition of oil-globules, and sclerosed patches are found in old cases.

If the inflammatory action be very severe, the nerve will be found to be completely destroyed by sloughing. The nerve may be found to be the seat of enlargements, which are to be seen at different localities in its course, and at each of these points there may be a different kind of change. Inflammation of a nerve-trunk, as I have said, is first attended by sensory changes, which may be local, or in other parts; as the result of reflected irritability; afterwards trophic changes may result either from the production of some pressure upon other parts, or through loss of function of the nerve itself.

**Diagnosis.**—The limitation of the pain, its aggravation by pressure, its constancy, and its character, enable us to generally distinguish it from neuralgia. In chronic neuritis it is not so easy to make such a diagnosis. The painful points found in neuralgia may be mistaken for the sensitive spots in neuritis. I have seen very few cases in which the pain of neuritis was not constant, and this is not the case in neuralgia, which is essentially



a paroxysmal disease. Painful swelling of the nerve and paralysis of muscles supplied are also evidences of neuritis, which will aid us in discovering the nature of the affection.

Muscular rheumatism has been spoken of by Erb as a condition with which the disease under consideration may be confounded. I consider such a distinction to be a refinement of diagnosis which cannot be made. "Muscular rheumatism" is, after all, a low grade of diffused neuritis, and the most we can do is to discover the cause of such pain.

Erysipelas, thrombosis, and embolism are distinguished by the evidences of subcutaneous swelling, oedema, etc., and by their somewhat diffuse character.

The presence of a traumatism should be taken into account, and its nature investigated.

**Prognosis.**—Structural alteration of a nerve must follow an inflammation such as has been described, and unless the symptoms have been very slight, there is a tendency to continuance, so that an attack of acute neuritis assumes a chronic character. If the inflammation has advanced centrally, so that a new plexus is involved, the prognosis is very bad. Treatment has much to do in some cases with prognosis.

**Treatment.**—To Mitchell we are indebted for excellent directions for the management of neuritis. He tried elevation of the leg or arm while bladders of ice were applied to every part of the limb, and  $\frac{1}{15}$  gr. hypodermic doses of atropia, with  $\frac{1}{2}$  gr. doses of sulph. of morphia, were injected every four hours, or oftener. He has used leeches, so that considerable local abstraction of blood should take place. Perfect quiet is highly important, and he recommends splints for the purpose. I have used the plaster bandage in a way to leave the course of the painful nerve exposed. The actual cautery is invaluable, especially when the disease is chronic, and it should be freely applied along the painful tract. Faradization does good, but I have no faith in the galvanic current, which only increases the pain. Hypodermics, either of morphia, atropia, or ergotine, in the neighborhood of the painful point, may be continued for some time, with the effect of diminishing the pain and the violence of the inflammation. Large doses of iodide of potassium are of especial service; and I have lately recommended inunctions of mercurial ointment with excellent results. This latter treatment is that which we are to employ when syphilis is suspected; and the good effects are sometimes seen in a few days. As a *dernier ressort* nerve-section may be tried; but if the neuritis has involved the nerve-plexus it does no good. It is only when a peripheral nerve is affected that it removes the disease.

In nerve-stretching—an extremely valuable surgical procedure—we possess a means which promises to be of great service. The nerve is exposed, and forcibly pulled, so that the limb shall be raised. In one instance the portion of the lower extremity, including the leg and foot, was drawn up by the sciatic, which had been bared in its course down the thigh.

## ANÆSTHESIA.

**Symptoms.**—An impairment or loss of cutaneous or muscular sensibility, either localized or extensive, may be the result of central disease, or it may be of a strictly peripheral nature. It is of the latter form that I now propose to speak.

The anæsthesia may imply loss of the sense of appreciation of extremes of temperature, contact, or painful impressions.

In the optic nerve, amaurosis is a result, and with this there is commonly anæsthesia of the ciliary nerve, so that the influence of light possesses no irritant effect. Deafness follows auditory anæsthesia, and loss of taste, anæsthesia of the lingual nerve.

Anæsthesia and analgesia may exist alone or in complication, and we are constantly reminded of this state in cases where operations are performed on insensible parts, the individual only feeling the power of traction or the contact of the surgical instrument. This is often observed in some of the uterine operations; and Dieffenbach<sup>1</sup> alludes to the anæsthetic condition produced by some of the agents employed, which only blunt sensibility, while the sense of contact still is preserved. I have myself witnessed this phenomenon in patients in whom local anæsthesia had been used.

In regard to the measurement of sensibility, and its impairment by disease, I may state upon the authority of Rosenthal,<sup>2</sup> that the sensibility to tickling is the first to disappear, then to contact and pressure, and temperature, and finally to pain.

In cutaneous anæsthesia a warm or cold body is not appreciable as such, but the individual can sometimes tell its shape, or feel the pressure made. A lump of ice is said to be irregular. The button of the heated cautery iron, if pressed against the skin, produces no discomfort, but only a sense of weight. The loss of tactile sensibility is generally abolished however, or greatly diminished. The patient will either not feel the points of the æsthesiometer at all, or, if he does, will be unable to tell how far they are separated.

The local temperature and vascular supply are altered in many cases, so that the warmth of the spot which has become anæsthetic is a degree or two below that of the sound parts adjacent. The vascular alterations are attended by bloodlessness and whiteness of the affected region. This diminished blood-supply of course invites pathological alterations of nutrition, for, when subjected to influences of temperature or injury which other normal districts would bear without damage, the anæsthetic skin becomes rapidly altered. Romberg<sup>3</sup> alludes to the occurrence of blisters and ulcerations which were readily caused during cold weather;

<sup>1</sup> Der Äther gegen den Schmerz, 1847, p. 61.

<sup>2</sup> Clinical Treatise upon Diseases of the Nervous System. Am. Translation, p. 173.

<sup>3</sup> Manual of the Nervous Diseases of Man, p. 202.

and I have repeatedly seen the effects of injurious pressure, of surgical operations, and of the application of irritants. In one patient brought to me I was surprised to find an extensive ulceration of the skin of the forearm, which had resulted from the use of a stimulating liniment which the patient had used with the idea of improving an anæsthetic state dependent upon rheumatism.

*Anæsthesia of the Fifth Pair.*—This form of anæsthesia is commonly of peripheral origin, and of thirty-five cases collected by Ortel-Ebrard<sup>1</sup> it resulted but nine times from intracranial tumors. It is of spontaneous origin usually; and the upper branch is most profoundly affected, so that the loss of sensibility is limited to the brow and region about the eye, by anæsthesia of the cornea, and consequent nutritive changes in that part of the optical apparatus. A case of this kind was reported by Dr. H. D. Noyes,<sup>2</sup> of New York, in which there was very decided sloughing of the cornea. The phenomena following anæsthesia of this nerve may be thus tabulated:—

|   |   |
|---|---|
| Involvement of ophthalmic branch.         | { Anæsthesia of upper eyelid and forehead. Irritating substances are not felt.  |
| Involvement of superior maxillary branch. | { Anæsthesia of middle portion of face. Insensibility of gums of upper jaw.   |
| Involvement of inferior maxillary branch. | { Anæsthesia of skin of lower portion of face; increased flow of saliva; mastication difficult; gums of lower jaw insensible. |

The patient sometimes finds that the edge of the tumbler or vessel from which he drinks occasionally feels as if it were broken. Several of these cases are reported by Bell.<sup>3</sup> In one of my cases the patient could not spit in a straight line, while the secretion of saliva was quite abundant. This same patient complained that his gums were insensitive. These peculiar buccal and labial symptoms are generally early and prominent expressions. Sense of smell and sensibility of the nostrils and tongue are lost when other branches are affected. A kind of anæsthesia, alluded to by Besnier, Rendu and others is that dependent upon venereal excesses and the pathological state is probably a lively spinal congestion. In a case reported by Besnier, there was some slight paresis of the lower extremities with analgesia, and pronounced loss of tactile sensibility. The patient was able to perceive temperature fluctuations. A cure followed six weeks of energetic treatment.

<sup>1</sup> Paralyse du Trijemeau, Thèse Paris, 1867.

<sup>2</sup> N. Y. Medical Journal, 1871.

<sup>3</sup> The Nervous System, etc., 3d ed., p. 333, *et seq.*



When the *radial* nerve is the seat of the peripheral trouble, it will be found that the back of the hand retains its sensibility. The lower extremities may be affected when the condition is the result of pressure made upon the sciatic, and in the case of several skin-diseases the loss of sensibility may be general. Leprosy, syphilitic alopecia, and other skin-diseases may all be attended by loss of cutaneous sensation, which is the result of local dermal alteration of function. Bulkley<sup>1</sup> has very ably considered this subject.

In this connection it will not be amiss to refer to a form of anæsthesia, called by Raynaud "*asphyxie locale des extrémités*," which is commonly described as a vaso motor disorder. Nine years ago I presented cases, and Dr. M'Bride has since discussed the subject in a paper read before the Neurological Society. Through contraction of the arterioles, the fingers become pale, and there is a sharply defined local syncope. The fingers are anæsthetic, and the sense of appreciation of temperature is lost. The arterial contraction may be the consequence of a temporary spasm, or it may have a grave permanency, and be followed by gangrene. The cases I have seen have been of short duration, and the subjects were women. The local syncope and anæsthesia is generally bilateral.

The anæsthesia often remaining after diphtheria is one of considerable interest. It may, or not, be associated with paresis, but in either case the velum palati is commonly affected, and in many patients other parts of the body become anæsthetic. See<sup>2</sup> reports an example in which the entire surface of the body was insensitive, the plantar surfaces even being affected, and, as a consequence, there was inco-ordination. This suggests the query whether the cases reported as locomotor ataxia of diphtheritic origin were not, after all, examples of plantar anæsthesia.

**Causes.**—Cutaneous anæsthesia may be due to pressure made upon, a nerve-trunk in its course, or to the compression of peripheral areas of greater or less extent, or to local impairment of function by exposure to cold, to certain chemicals, or to like agents; while general diseases, such as syphilis or rheumatism, by local disease and infiltration, greatly alter the function of cutaneous nerve-filaments. The toxic effects of lead shown in abolition of cutaneous sensibility were pointed out by Beau<sup>3</sup> in 1848. In 38 cases analyzed by him, loss of tactile sensibility was detected not only in skin of the forearm and arm, but in parts lined with mucous membrane, the pharynx and the interior of the nose. Intense cold, liniments which contain aconite, or long immersion of the hands in fluid of any kind, will result in a loss of sensibility. One of my patients was a dyer, whose hands were kept in warm dye-liquids for many hours; and some of the French writers give examples of the disease among

<sup>1</sup> The Relations of the Nervous System to Diseases of the Skin. Archiv. of Elect. and Neurology, 1874-5.

<sup>2</sup> Gaz. Med. de Paris, 1864.

<sup>3</sup> Recherches sur l'anesthésie, Archives. Gen. de Med., 1848.

washerwomen. Alkaline fluids are more favorable to its production than any others. Tight splints, blows; diphtheria and other acute maladies, hysteria, and several other conditions play a part in its etiology.

**Diagnosis.**—Peripheral anæsthesia must be diagnosed from the central condition, and it is necessary that we should bear in mind not only the anatomical arrangement of the nervous supply, but the coexistence or absence of symptoms of central disturbance. Among the latter are loss of power, which usually accompanies the anæsthesia, or one or more of the many symptoms previously alluded to.

Trigeminal anæsthesia is, perhaps, more difficult to trace out than that of other nerves. Romberg<sup>1</sup> thus enumerates the indications of anæsthesia of peripheral or central origin:—

“*a.* The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

“*b.* If the loss of sensation affects a portion of the facial surface, together with the corresponding facial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

“*c.* When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

“*d.* If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain.”

**Prognosis.**—It is by no means bad after the cause is removed. Anæsthesia from pressure is rapidly restored, provided the mechanical injury be not too great. If there be division of the nerve, the process of reparation, which rarely extends for more than a few months, is followed by a healthy return. With syphilis and metallic poisoning, and skin diseases the case is different.

**Treatment.**—Electricity offers the best mode of relief. The wire brush and faradic current are to be employed every day; and at the same time applications of alternate heat and cold, friction and massage, are useful adjuvants.

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<sup>1</sup> Romberg. A Manual of the Nervous Diseases of Man. Sydenham trans., vol. i. p. 213, *et seq.*

## TUMORS OF NERVES.

**Synonym.**—Neuromata.

A nerve may be the seat of either a syphilitic, cancerous, sarcomatous, myxomatous, or other growth which may involve or destroy some point in its continuity, or form as a benignant tumor at its point of severance.

Very little has been written on this important subject; but among the most valuable contributions to the literature of nerve-tumors is an excellent thesis by Foucault,<sup>1</sup> and various scattered articles by Verneuil,<sup>2</sup> Le Fort, Axenfeld, Roger, and others.

Nerve-tumors may be classified as *neuromata* (*nervous neuroma* of Weber) and *medullary* nerve-tumors, which involve the nervous structure itself; and *pseudo-neuromata*, which include the fibromata, myxomata, epithelioma, as well as cysts and tumors of a like character.

Medullary or ganglion tumors are quite rare, and are of a hyperplastic character. Lebert<sup>3</sup> described a neuroma of the superior cervical ganglion, in which all traces of true nervous matter had disappeared, and naught remained but a fibro-fatty structure. Robin<sup>4</sup> has found a neuroma in the solar plexus, and Virchow has also brought forward examples.

Neuroma of nervous fasciculi (*nevrômes fasciculés*) include the little painful tumors which are met with after amputation, which give rise to stump neuralgia, and attain the size often of a hazel-nut. Dupuytren,<sup>5</sup> Cornil<sup>6</sup> and Ranvier, Axmann<sup>7</sup> and Weissman,<sup>8</sup> have all described their appearance and structure, which is fibrous and hard, and the nerve tubes are tortuous and interlaced.

The pseudo-neuromata are of many varieties. They are developed usually in the course of the nerve, and the neurilemma is thickened, and should the nerve be cut across, a white or yellowish hardening will be presented. Should the tumor be fibrous, the peculiar microscopical appearance may be observed. Fibromata rarely exceed the size of an almond; but when there is any fluid found, as in the case of fibro-cystic tumors, the volume of the enlargement may be much greater.

The accompanying cut represents a sarcoma of the ulnar nerve, and was observed by Demarquay at the Maison Municipale de Santé.

Nerve-tumors prefer the nerves of the upper and lower extremities, and in the leg the posterior tibial nerve seems to be a common site. It is not uncommon to find a great many tumors of this kind existing at the same time. In one case reported by Foucault, 1400 of them were found, but

<sup>1</sup> Sur les Tumeurs des Nerves Mixtes, Thèse de Paris, 1872.

<sup>2</sup> Arch. de Méd., tome xviii. 1861.

<sup>3</sup> Mém. de la Soc. de Clin. 1853, 3 fasc.

<sup>4</sup> Comptes Rendus de la Soc. de Biol., 1854.

<sup>5</sup> Loc. cit.

<sup>6</sup> Mémoires de la Soc. Biologie, t. v., 3d série, 1863.

<sup>7</sup> Beiträge zur. mikr. Anat. du Gänglion Nervensystems, Berlin, 1853.

<sup>8</sup> Ueber Nervenbildung (Zeitschr. f. Rationelle Med. 1859.)



this is exceptional, and it is probable that multiple neuromata are more frequently found in patients who are of the cancerous, syphilitic, or some other diathesis. Very often these growths, the result of injury, are subcutaneous. In one of my cases the growth was found at the elbow at the exposed site of the ulnar nerve, and its origin followed a blow upon that part.

Pain, as I have said, is the prominent symptom of such growths. This pain may appear upon the tumor, but usually follows its establishment. It may be localized or diffused, or may be provoked by pressure on the spot or spots which mark the site of the growth; for, when the tumors are multiple, of course the sensory troubles are equally numerous. The pain may radiate from the tumor, or may dart down or up the affected nerve. It is not so intense with fibromata, syphilomata, or sarcomata, or when the tumor is composed mainly of true nervous tissue, as is the case in stump growths, and in these examples it is productive of severe neuralgia of a reflex character. Spasms, permanent muscular contractions, and sometimes a peculiar constriction of the thorax of a tetanic nature, with epileptiform seizure and centripetal pain, are indicative of certain reflex disturbances.

**Treatment.**—Operation seems to offer the only hope of relief, and in stump neuromata re-amputation is oftentimes necessary. It will be found necessary to deeply anæsthetize the patient, as the sensibility is so morbidly active that ordinary anæsthesia is insufficient. The removal of a considerable piece of the nerve is advisable, for it is not rare to find considerable infiltration or deposit in its substance for some distance from the actual growth. In syphilis, mercurials and the iodides offer some show of relief, and these are the only remedies when the growth is deep-seated. Legrand<sup>1</sup> and others have recommended caustic applications in superficial regions, and Siebold père removed a tumor in this way from the anterior tibial nerve. The operation is rather severe, and is attended with doubtful success.

Fig. 69.



Sarcomatous Neuroma. (Foucault)

<sup>1</sup> Gaz. Méd., Compte-Rendus de l'Acad. des Sciences, 1858.

## CHAPTER XVIII.

## DISEASES OF THE PERIPHERAL NERVES (CONTINUED).

## LOCAL PARALYSES.

## FACIAL PARALYSIS.

**Synonyms.**—Bell's paralysis; Histrionic paralysis.

Facial paralysis may be either double or single, but is more often the latter; and it may depend upon a lesion of a peripheral kind, or one that may be seated in the temporal bone, or at any point in its course within the cranial cavity, or else at its origin.

The bilateral form is rare, and is always the result of a central lesion; but the peripheral form is unilateral, and is a very common affection.

**Symptoms.**—The patient, after exposure, may suddenly be attacked; and the first intimation he generally has is in the morning, when he arises. He then finds his face to be all awry, and his appearance is absurd to the last degree; one side being drawn up, while the other is immobile, as the muscles of expression are powerless. If he laughs, the contortion is more marked, and if he attempts to whistle he will find that he is utterly unable to do so. The corner of the mouth on the sound side is drawn up, and the furrow at the angle of the nose is more marked than natural. The opposite side of the face is smooth; and, in the passive state, the muscles seem to sag heavily downwards. It is impossible for him to corrugate his eyebrows; and the eyelids of the paralyzed side cannot be closed, so that dust and foreign substances collect, producing irritation and discomfort. This is due to the paralysis of the orbicularis, and at the same time the levator palpebrarum contracts and keeps the eyeball exposed. The individual is unable to blow out a candle, and articulation is interfered with to a slight degree. Should he be an old man, any wrinkles or furrows that may have existed on the paralyzed side are effectually effaced, and give that part a most ghastly appearance. Considerable discomfort results from the insufficiency of the lower lid, so that the tears, instead of being conducted to the lachrymal canal, find their way over the cheek.

If the lesion be situated within the temporal bone or the cranium, a much more extensive paralysis may result. This is indicated by a loss of power of the muscles of the palate, uvula, and other parts of the fauces.

When the patient opens his mouth, the palatine arch will be found to be altered, the anterior pillars of the fauces being shorter, so that one side

falls lower than the other.<sup>1</sup> The uvula will also be found to be arched, the concavity looking towards the sound side. The tongue will then also be paralyzed, so that its surface is smooth; and there may be a dryness of the mouth, which results from diminished secretion of saliva. Should the portio mollis be affected, there may be, in addition, deafness. If the third nerve be affected, as it sometimes is, of course ptosis with dilated pupil and paralysis of the recti will result.

Roux,<sup>2</sup> who was paralyzed in this manner, perceived a metallic taste in the right side of the tongue.

Should the paralysis be bilateral, the patient's features will denote an entire lack of expression, and there is not the slightest evidence of any emotional excitement expressed, even should the patient be agitated by the most intense pleasure or the deepest grief. The muscles are flabby, and the face seems more like a mask than what it really is; and, as is the case in advanced progressive muscular atrophy, the only animated features are the eyes.

Romberg<sup>3</sup> describes the appearance of a patient in these words: "In a girl of 16, in Dupuytren's Clinique, who was affected with bilateral paralysis, there was no distortion, but a pendulousness and entire absence of motion was perceptible in all the features. The eyelids only closed half, the lips stood apart, and played backwards and forwards from the impulse of respiration. The expressive countenance bore a serious character, which contrasted forcibly with the patient's frame of mind. She was heard to laugh aloud, but the laugh appeared to come from behind a mask." Sensation is not usually impaired.

**Causes.**—The peripheral form of paralysis may follow exposure to cold, rheumatic exudation, and injuries of various kinds. A cause which is frequently observed is the chilling of the face by a blast of cold wind; and the frequency of this cause has led to the adoption by the French writers of the term, "*Coup de vent*." I have met with many cases in which the paralysis took place after a railroad journey, the individual having sat by an open window.

In one instance the patient, who was a young lady, had been dancing violently, and had afterwards gone into a damp conservatory to cool off; the palsy was shortly afterwards noticed.

Rheumatic exudations may produce pressure upon some of the nerve-twigs, or an attack of parotitis may result in pressure upon the cervico-

<sup>1</sup> Hughlings Jackson (*London Lancet*, Jan. 16, 1875) does not consider that deviation of the palate occurs in uncomplicated disease of the portio dura, and he does not believe deviation of the uvula to be uncommon in healthy people. Troltsch says that the levator palati is supplied by the vagus, which explains the phenomena witnessed by Jackson, viz., marked palsy of one side of the palate, with palsy of the vocal cord on the same side, as a result of intracranial disease. This case, however, is exceptional.

<sup>2</sup> Descot. *Diss. sur les Affections locales des Nerfs*, Paris, 1825, p. 331.

<sup>3</sup> *Op. cit.*, vol. ii. p. 268.



facial branch. Injuries of the nerve, whether such as follow coarse traumatism or accidental section of the nerve during a surgical operation, are sometimes the cause of the paralysis.

Weir Mitchell relates several cases of this kind. Three of these (Cases 61, 62, and 63) followed gunshot wounds.<sup>1</sup> In one the portio dura of the left side was injured, and as a consequence there were facial palsy, impaired speech, and loss of gustation. Hearing was impaired from shock transmitted to the auditory nerve. Sir Charles Bell<sup>2</sup> divided the facial in removing a tumor, and other cases are reported by various surgeons.

Carious disease, as well as fractures of the temporal bone, often produces paralysis, either by pressure, by the products of inflammation, or by direct contusion.

Tumors and various aural growths are occasionally causes of this second form of facial palsy; and Romberg<sup>3</sup> reports a case, seen by Henle, in which a tuberculous deposit was found beneath the middle lobe of the brain, with destruction of the petrous portion of the temporal bone; and Froriep<sup>4</sup> also found a deposit of tuberculous matter in the Fallopian canal, with caries of the petrous portion of the bone.

Degeneration, exudation, and tumor in or near the pons may also be the cause of the deep form.

The following case is an example of deep-seated paralysis, evidently dependent upon aural disease:—

Samuel M., aged 27; United States, canal boatman; came to me July 3, 1876. Three days before the first visit, after exposure while washing the decks of his boat, he became paralyzed. He had had earache before for several days, but did not consider it of sufficient moment to quit work; and his first intimation of trouble was the discomfort produced by particles of dust which blew in his eye. He could not close his left eye, and on looking in the glass he discovered the paralysis. There was no pain, nor any subjective sensation of any kind. He found that he could not laugh, nor blow his nose, and when he attempted the latter "the wind came out of his mouth." When I saw him there was paralysis of both branches of the seventh nerve. Hearing was very imperfect, and he could not count the ticks when the watch was pressed to the left ear. The left palatine arch was obliterated, and he could not fully protrude the tongue, which was quite dry. The left side of the face is quite flat, and the muscles of the other side act to such a degree as to draw up the right corner of the mouth, producing the characteristic deformity. When he opens his mouth the orifice is unsymmetrical. He cannot whistle or expectorate, he cannot close the left eye, but when he attempts to do so the ball is drawn upwards, so that the sclerotic is shown to a great extent. Contractility to both currents fair; mediate and immediate galvanization are followed by muscular response. He has some earache. When the elec-

<sup>1</sup> Injuries of Nerves, etc., p. 392, *et seq.*

<sup>2</sup> The Nervous System of the Human Body, 3d ed., 1836, p. 56.

<sup>3</sup> Romberg, *op. cit.*, p. 272.

<sup>4</sup> Massalien, *Diss. Inaugur. de Nervo Faciali*, Berolini, 1836.

trode is passed over the superficial points of the fifth, there is decided pain, no anæsthesia; force of masseter muscles tested by putting the dynamometer bulb between the teeth and interposing two pieces of wood; no loss of power as compared with my own attempts. Tympanum congested; and I infer that there is middle ear disease. R. Potass. iodid. and syringing ear with warm water.

*July 6.* Has had intense pain in the left ear, throbbing and pains which radiate over the head. Pressure over mastoid process gives great distress, as does electrization. Leeching to inner tragus.

*9th.* Says that there was a discharge of pus last night. After syringing out I find a perforated tympanum. Stopped iodide, and ordered syringing with warm water and glycerin.

*13th.* Discharge from ear much less. Used iodoform powder locally. Muscles do not respond so well to either current. Iodide renewed.

*17th.* No response to current. Faradized nevertheless.

*19th, 21st, 23d, 27th.* Used iodoform. Aural disease almost well, but patient still deaf. Muscles still inactive.

*30th.* Tested sense of taste, and find it markedly affected; his tongue seems quite smooth. He has had from the first some clumsiness in speech.

*Oct. 1877.* There has been very slight improvement since the last entry. The facial deformity is not so great. He is still deaf. His speech is clear, but he cannot whistle as yet. The muscles do not respond to the currents. He suffers great annoyance from the accumulation of saliva, and when he expectorates he soils his clothing.

**Pathology.**—The anatomical distribution of the facial nerve, and its connection with other nerves may be referred to in illustration of the pathology of the affection. Beginning externally, we find that the facial nerve supplies the muscles of the face, the *malar branches* innervating the orbicular muscles of the eyes; that the *infra-orbital* supply the buccinator and orbicularis muscles, and the levator labii superioris *alæque nasi* muscles; while the *cervico-facial* division of the nerve passes through the parotid gland, and supplies the muscles of the mouth and lower jaw; consequently a lesion of any of these branches, or of the main trunk at its exit from the stylo-mastoid foramen would be followed simply by paresis of the facial muscles. Should the lesion take place in the aqueductus Fallopii, or behind the geniculate ganglion, we would find as a consequence paralysis of the muscles of the face, the tongue, through paralysis of the *chorda tympani*, and paralysis of the palate muscles, through paralysis of the larger *superficial petrosal nerve*, which runs from the geniculate ganglion to the spheno-palatine ganglion. Deep lesions may involve the third nerve, and perhaps the sixth. The lesions and their results may be thus arranged:—

*Paralysis of the Seventh Nerve.*

| EXTERNAL THIRD.  | MIDDLE THIRD.  | INTERNAL THIRD.  |
|--|--|--|
| Facial Branches.<br>——   | Petrostalar nerves, Auditory<br>(Portio mollis), Chorda<br>· Tympani.<br>——  | Possibly lesion involve<br>the 3rd and 6th nerves, and<br>then besides all of the fore-<br>going there may be paraly-<br>sis of the levator palpebræ<br>and the recti muscles. |
| Paralysis of the<br>Orbicularis palpebrarum,<br>Corrugator supercilii,<br>Levator labii, etc.,<br>Pyramidalis nasi,<br>Diagastic,.<br>Buccinator,<br>Orbicularis oris,<br>Depressor anguli oris,<br>Levator labii inf. | Paralysis of all the fore-<br>going as well as lingualis,<br>tensor and laxator tym-<br>pani, levator palati, and<br>azygos uvulæ. |  |

**Diagnosis.**—The appearance of facial paralysis may be a source of alarm to the individual, who is ready to believe it a feature of cerebral hemorrhage or deep organic trouble. It is much more profound, however, than the form which accompanies cerebral hemorrhage; and generally there is hemiplegia of the extremities in the latter disease. In this form it is impossible for the patient to shut the affected eye, while in the other disease there is usually no difficulty in so doing. Sensation is also affected in the paralysis from cerebral hemorrhage, and it is not unusual to find ptosis. The matter of importance, however, is the diagnosis of the variety of facial palsy, superficial or deep: and we may avail ourselves of electricity in settling this point.

If the paralysis be peripheral, the muscles retain their contractility for several weeks. If, on the contrary, the lesion be central, or in a nerve-trunk, they lose their power of response to a faradic current in a few days, and later to even a galvanic current, and the muscles finally become atrophied. If the paralysis be due to bulbar disease, the appearance of symptoms indicating impairment of other nerves and an eventful fatal termination should settle the nature of the affection, and enable us to make a prognosis. The existence of carious disease and its indications, the complication of deafness, and the co-existence of indications of deep trouble, should be all taken into account.

**Prognosis.**—The prognosis of the peripheral form of the disease is very good, and under proper treatment the paralyzed muscles may be rapidly restored. There is generally early loss of muscular contractility, which only the galvanic current can restore. If there is no response to electrical excitement, and the muscles of the paralyzed side are wasted and contracted, there is little to be hoped for. I consider that more depends upon the early adoption of electrical treatment than anything else; and if there be a delay in the selection of remedies, and in the attempts to restore the muscles by mechanical support and electricity, the prognosis, which may have been favorable in the beginning, becomes less and less so, the longer action is delayed.



Syphilis is a favorable element if the paralysis be due to deep lesions; but, if it be caused by brain-tumors, exudations, or degeneration, there is scarcely any hope.

**Treatment.**—It is necessary in this disease to direct the treatment not only to the cause, when one can be found, but also to the restoration of the paralyzed muscles.

Should rheumatism exist, we are to employ colchicum and iodide of potassium; if syphilis, the specifics which are at our disposal; and if there be caries, we are to improve the patient's general health by nourishment and stimulants, and to apply such local treatment as may seem proper. The medicaments which will be found to be of service for the direct treatment of the paralysis are strychnia, iron, and quinine. Electricity is of great service; and we may begin with the galvanic current and use the faradic as soon as it can produce contractions. The *negative* pole of the galvanic battery should be placed behind the ear, and the positive pole passed over the different facial muscles. The glass "bain électrique" should be applied to the eye, so that the orbicularis shall be brought under the influence of the current.

The mechanical treatment of facial paralysis has been advocated by Detmold, and with admirable results. A piece of tin wire is bent at both ends (Fig. 70), and one end is passed over the ear and the other hooked in the angle of the mouth, so that the muscles of the paralyzed side shall be supported. In several of Detmold's cases it was found to work exceedingly well.

Fig. 70.



Wire Hook for the Treatment of Facial Paralysis.

This apparatus may be worn at night or during the day, and does not give the patient any discomfort whatever.

Dr. Van Bibber has suggested, in the treatment of ptosis, the use of a small strip of court plaster, which is affixed to the upper lid and to the forehead above.

I may append a case of facial palsy of a syphilitic nature cured by electricity in a remarkably short space of time.

W. O. I., 30 years; United States, boatman. *Previous history:* He has never been seriously ill, but ten years ago he had a chancre, followed by marked secondary symptoms. The only other ailment was a severe attack of rheumatism, occurring a year before. This was undoubtedly a secondary symptom. His present difficulty began three months ago. At

night he was disturbed by intense cephalic pains, dizziness, and disordered vision. For several days the pains were steady and most violent under either temple; he was also annoyed by post-aural pains. He then found that his hearing was becoming less acute, till the lesion finally occurred. This took place toward the latter part of July, 1880. He awoke in the morning and felt a pain in the head, attended by swelling and puffiness in the face. His attention was called by several of his associates to the "crookedness" of his face. He looked in the glass, and saw the drooping of the left side of the face, with complete paralysis of the muscles at the corner of the mouth; then followed total loss of hearing, and he could not appreciate the loudest noises when the sound ear was closed. The paralysis increased every day.

A few days after this the eyelid drooped, and he found it impossible to open or completely shut the eye. It became congested and irritated, and he experienced a burning sensation with photophobia. His condition grew gradually worse, till he was compelled to leave his employment and seek medical aid. He never had had otorrhœa or ear affections of any kind, nor had been paralyzed. His habits were good, and his hereditary history favorable. When he applied to me, I found paralysis of the entire seventh nerve, motor ocularis, and disturbance of the sympathetic of the eye. There was no appreciable power in the orbicularis oris, levator labii superioris et alæque nasi, or other muscles. He could hardly insert the finger in the mouth without pulling down the jaw with the other hand. He experienced mastication and deglutition from involvement of the left side of the tongue, which, when protruded, inclined to the right side. With this there was indistinct articulation, and I was led to infer paralysis of the lingualis muscle. From the patient's previous history I was led to suppose that syphilis was the primary cause of the trouble, and, from the depth of the lesion, that the seventh nerve was paralyzed at a point above its division. From the specific features of his case I deemed the iodide of potassium to be the best remedy, and he was therefore put upon grs. v thrice daily. Hypodermic injections of strychnia and atropia did much good in relieving the severe cephalalgia. Localized galvanization was resorted to, and both the primary and secondary currents used. After the nerve and its branches had been pencilled over with stick caustic, one electrode was applied to the ramifications of the nerve, while the other was placed over the mastoid process. So successful was this treatment that after a daily séance lasting twenty minutes, in three weeks the patient's face was much more symmetrical, and the act of mastication improved. The pains like wise disappeared under the same current. Occasional directions of this and the faradic current over the eyelid did much toward the improvement of sight.

It now occurred to me that Matteucci's experiment on the ear might be followed by gratifying results; so its cavity was filled with water, and one of the battery-wires, finely covered with sponge, was gently introduced into the external meatus. After four weeks his hearing was so markedly improved that he easily distinguished loud voices when the sound ear was closed.

*November 12* (seven weeks after commencement of treatment). During the application of the current the face resumed its expression, and he was able to close his eye completely. He is greatly improved; injections discontinued. He has almost complete control over the levator palpebræ—this is marked in the morning; articulation good.

28th. Has now taken the battery for nearly ten weeks, and is about to discontinue treatment. The face is perfectly symmetrical, and the hearing nearly as perfect as ever. The only remaining disfigurement is a slight drooping of the eyelid on the affected side; appetite good, and, though emaciated at first, he has now completely regained his former healthy condition.

### TRAUMATIC PARALYSIS.

Under this head I propose to speak of those forms of lost power dependent upon partial or complete nerve-section, or pressure made upon a nerve in its course, such as is often seen in a familiar form known as decubitus paralysis, as well as in the loss of motility produced by cold or other influences which may affect the ramifications at the peripheral end of a nerve-trunk. There is no regularity either in the form of invasion, the extent of the paralysis, or its locality. Suffice it to say, that both upper and lower extremities may be affected, the upper especially, and that such paralysis is not bilateral. The liability of the upper extremities to this accident is probably explained by their use in many of the necessary actions of everyday life. These forms of paralysis may be divided into three groups: (1) Paralysis following section or destruction of a nerve-trunk or its branches; (2) Paralysis following pressure; (3) Paralysis following cold, or general disease.

*Division of a Nerve-trunk.*—If the section be complete, the paralysis will be equally complete and immediate. There is likely to be, in addition to lost sensation and motion in the muscle supplied by the nerve, various trophic defects, which may consist in exfoliation of the skin, and in changes in the condition of the nails, which become curved, crenated, and deformed; and sometimes eruptions. The loss of motion, of course, will depend upon the importance of the group of muscles supplied by the nerve; and it does not follow, by any means, that the member is utterly useless, as some muscles may escape the paralysis. Should supuration and inflammation occur at the wound, there may be various disturbances of sensation, and also lowered temperature in the paralyzed side.

*Contusions and Punctured Wounds.*—The injuries produced by kicks, or direct violence, when the skin is not broken, are very commonly followed by traumatic paralysis. These are likely to occur when the nerve rests upon some bony prominence, and when there is no muscular or other cushion to make the blow less slight. I can recall cases of this kind, one in particular, where the individual fell in the street, striking his elbow upon a projecting stone. There were no immediate symptoms except a tingling and sharp pain, but in a few days there was loss of power, and some hyperæsthesia of the forearm.

The experience of surgeons furnishes us with numerous examples of peripheral paralysis from dislocation. Dr. S. G. Webber,<sup>1</sup> of Boston, has brought forward several very interesting cases of this variety, with dislo-

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<sup>1</sup> Boston Med. and Surg. Journal, Dec. 18, 1873.



cation of the humerus; and Onimus and Legros<sup>1</sup> a case which Webber presents in his article to illustrate a form of paralysis following dislocation of the femur:—

“A man, forty-six years of age, suffered an ilio-ischiatic dislocation of the femur, which was produced by violence exerted by falling rocks and earth. Severe pain, anæsthesia, and immobility of the leg existed at first, but the pain subsequently disappeared, and the anæsthesia remained. After an attack of facial erysipelas the pain in the legs returned. Five months later the left leg was found to be cold and smaller than the other, and œdematous about the tibio-tarsal joint. The leg could be flexed and raised, but the foot could not be raised nor the toes extended. Sensation was diminished, as was electro-muscular contractility, especially in the flexors and extensors of the leg, the muscles of the calf and the peronei, as well as the tibialis anticus and extensor communis.”

In Webber's case of paralysis following dislocation of the humerus, the biceps and deltoid were most affected, and there was anæsthesia over the deltoid.

J. S. came to the N. Y. State Hospital for Disease of the Nervous System, June 9, 1871, with the following history: During an altercation with a fellow-laborer he was thrown off a scaffold, and dragged by his right arm for some distance. When he arose he found that the whole arm was very painful, and a few mornings afterwards the right wrist became very weak, and he was unable to grasp any object or move his fingers. Sensation was unimpaired.

Nerve-injury following dislocation is not always the same, there being in some cases simply pressure, and in others rupture of the nerves by strain; and of course the prognosis depends much upon the fact whether there be simple contusion or actual laceration, as there was in a case reported by Hilton.

Pressure upon nerves may be made by the products of inflammation, cicatrices, callous tumors, or by improperly arranged splints, or the pressure of a crutch or some hard substance, or by the maintenance of a constrained position for an extended period. The products of a periostitis may exert pressure upon a nerve-trunk, or an exudation which makes compression either in its course or at its ramification, may either account for a paralysis. There is always some painful indication at first, and occasionally a neuritis, after which the loss of power takes place. Movement of the limb aggravates this pain, or pressure over the nerve has the same effect. Pressure from a cicatrix is quite rare, and it is only when very extensive contraction of the cicatrix occurs that any such condition of affairs can exist. So, too, is pressure from callus an uncommon cause of paralysis, and but a few cases of this kind have been mentioned.

The pressure of the nerve by a tumor may be first indicated by hyperæsthesia, and secondarily by loss of motion and sensation, and the duration of the first stage depends upon the site of the tumor, its rapidity of

<sup>1</sup> *Traité de l'Electricité Médicale*, Paris, 1872.

growth, and the room for increase in size. In certain situations where there are bony eminences or cavities, and where there is no room for expansion of the mass without consequent nerve-compression, the loss of function is very quickly produced.

By far the most familiar form of peripheral paralysis is that which follows the compression of nerves during the continued maintenance of a constrained position, the nerve-trunk being pressed against some bony eminence, or impinged upon by some tendon or muscular mass. The musculo-spiral nerve is, from its exposed position, most commonly affected. The common modes of onset may be the following: The patient falls asleep with his elbow resting upon some hard substance, and awakens to find his forearm devoid of power, so far as extension is concerned. There is some anæsthesia as well. The following are examples:—

M. P. went upon a spree, and when he became sober found his arm numb and cold, and devoid of power; muscles respond to faradic current; unable to force dynamometer column to 6.

T. W., four years ago, fell asleep with his left arm under his head; when he awoke his arm was numb and powerless. Soon after formication appeared. After seven months, pain, which subsequently became paroxysmal, began in the arm, coming on every two or three minutes. Response only to galvanic current.

In one case, reported by Webber, the paralysis was the result of carrying a basket of lemons, pressure being made on this nerve.

Mitchell<sup>1</sup> speaks of paralysis of this kind resulting from the most simple causes. In one case, that of a child, pressure was made by a string passing over the finger. And in other cases reported by Brinton,<sup>2</sup> it was found that the paralysis followed the rough use of a pair of cord handcuffs upon a prisoner who was being taken to the police station.

The use of the forceps is occasionally attended by paralysis of the facial nerves, the blades of the forceps making pressure upon the portia dura. In these cases there is paralysis of the facial muscles, an inability to nurse owing to the paralysis of the orbicularis oris, but no palatine loss of power, which serves to diagnose the effects from the form due to intracranial trouble. The mother may be paralyzed from pressure by the forceps exerted upon the pelvic nerves, but this accident is an extremely rare one.

Accumulation of feces produces paralysis generally by reflex irritation, and rarely by direct pressure. But few of such cases have been reported, and of these, one detailed by Portal<sup>3</sup> is of great interest, from the fact that spinal curvature favored the accumulation of feces and the exertion of pressure upon the nerves of the lumbar plexus.

<sup>1</sup> Op. cit., p. 126.

<sup>2</sup> U. S. San. Com. Reports.

<sup>3</sup> Cours d'Anatomie Médicale, t. iv. p. 276, quoted by Mitchell.

Cold or malaria may also be causes of a form of peripheral paralysis. In speaking of facial palsy I have alluded to the variety known as the "Coup de vent." This sudden origin from exposure to damp and wind is, however, much more rare than that which follows intense cold. I have had several cases of this latter kind among draymen, sailors, and others who have been obliged to work for a protracted period in an exposed place. There is at first a numbness, and afterwards a complete loss of power, which may be bilateral.

In peripheral paralysis there is a diminution of electro-muscular contractility after the first few days, and if there be complete section of the nerve this susceptibility to electric stimulation is lost, first to the faradic, and at the end of a week or two to galvanic stimulation. If a few fibres remain intact, it will be found that certain muscles are unaffected, and of course electrical irritation meets with a ready response. Changes of color in the paralyzed limbs are the rule, and there may be an extensive blanching or patches of discoloration dependent upon the irregular circulation. Analgesia and anæsthesia generally exist in some degree, while changes of temperature are not so readily perceived as on the sound side.

As the nerve is restored, electro-muscular contractility returns, and finally the patient is enabled to produce contraction at will.

Arlong and Tripier<sup>1</sup> have alluded to the rapid return of sensibility in distal parts after nerve section, and explain it by the theory that there are small communicating fibres between the severed portions, but this view has not been generally received. The expression of certain well-defined peripheral paralyses is anatomically the following:—

#### UPPER EXTREMITY.

*Paralysis of the Circumflex Nerve:* Loss of function of deltoid and teres minor muscles. The patient is consequently unable to put his hand to his head or raise it from his shoulder. The skin over the shoulder is anæsthetic.

*Paralysis of the Musculo-Spiral Nerve:* Loss of function of supinators and extensors. The loss of power is quite decided and there is some accompanying anæsthesia confined to the back of the forearm and a part of the hand. The extensor paralysis of the middle and index fingers is quite conspicuous.

*Paralysis of the Ulnar Nerve:* Loss of function of many of the important flexors, notably of the f. profundis and f. carpi ulnaris—shown in difficulty of flexing hand and little finger. Adduction is enfeebled. Sensation is blunted pretty much all over palmar surface; to a marked degree over thumb and over the two inner fingers and half of the third finger.

*Paralysis of the Median Nerve:* The patient presents chiefly evidence

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<sup>1</sup> Journal de l'Anatomie et Phys., etc., March and April, 1876.



of flexor paralysis, more profound than in last mentioned variety. The muscles of the ball of thumb are affected so that it is extended through antagonistic contraction of extensors. The palm of the hand and radial side of ring finger are anæsthetic. Through paralysis of the pronator radii teres he cannot pronate his hand.

#### LOWER EXTREMITIES.

Paralysis of the larger nerves does not commonly occur as a result of pressure or injury at a point in their course outside of the pelvis. Sciatica is occasionally attended by loss of motor power, and aggravated glandular disease may give rise to crural paralysis. Syphilitic infiltration may prove to be the origin of such trouble, or aneurismal swellings may be attended by the evidence of neural pressure. Pain and surface anæsthesia are associated with such paralyzes. Falls and blows upon the buttocks may give rise, in rare instances, to paralysis of the muscles of the thighs and buttocks, and Wilks speaks of the wasting of the glutei muscles as an evidence of loss of power and an accompaniment of certain neuralgic affections.

Paralysis of the nerves of the leg interest us much more, and as a consequence, we are furnished with weakness in the movements of the leg and foot. Peripheral paralysis resembling, in some respects, so far as the loss of power is concerned, certain spinal paralyzes of organic origin.

*Paralysis of the Peroneal Nerve:* Extensor paralysis of muscles supplied by its branches, viz.: External saphenous, musculo-cutaneous and anterior tibial. As a result, the muscles upon the anterior and outer part of the leg and toes are paralyzed with anæsthesia, chiefly of the integument covering the anterior part of the leg, and the inner side of the great and second, and the whole of the third and fourth toes, and the inner side of the little toe.

*Paralysis of the Posterior Tibial Nerve:* Loss of function of the posterior muscles of calf, and the flexors and abductors of toes. There is cutaneous anæsthesia of the plantar surface. The anæsthesia may be confined to the outer side of the fourth and little toes.

**Diagnosis and Prognosis.**—Progressive muscular atrophy and cerebral diseases are to be disposed of, and if we see the case after the onset we may be deceived. In the former it must be remembered that there are fibrillary contractions, and that the atrophy precedes the paralysis. The electro-muscular contractility is also preserved for some time.

In cerebral paralysis the electro-muscular contractility is preserved, and if anything exaggerated. Cerebral palsies do not involve such extensive sensory impairment. Spinal paralyzes are *usually* bilateral, a fact which distinguishes them from peripheral troubles.

Mitchell also alludes to the fact pointed out on a previous page, that in peripheral palsies there is none of the delay in transmission of impression which characterizes either spinal or cerebral trouble.

Westphal<sup>1</sup> has in reviewing an admirable article by Vulpian,<sup>2</sup> referred to the various interesting pathological changes which follow division of spinal nerves. His experiments were made to determine the muscle-changes which follow separation from the cord. His conclusions may be thus summed up:—

If a spinal nerve be cut through at any point between the spinal ganglion and the periphery, the nerve-fibres of the central portion undergo atrophy *en masse*, without their individual character being altered; but the peripheral part of the nerve-trunk undergoes what Vulpian calls “histopathic change,” *i. e.*, a breaking up or “splitting” of the medullary substance.

Atrophy of muscles follows section of a motor nerve; and, in addition to this, electric contractility is impaired.

The absence of central symptoms of any kind, the loss of both motion and sensation in a limited area, absence of reflex contractions when the sensory fibres are irritated, and voluntary motion lost, are evidences of the peripheral nature of these paralyses.

**Treatment.**—Traumatic paralysis, like the facial form, should be treated with an idea of removing the cause should it exist, and afterwards restoring the integrity of the nerve and muscles, and preventing muscular atrophy. If the nerve-trunk be severed, of course all we can do is to await the union of the divided ends. If a tumor makes the destructive pressure, it should be removed if possible. It is hardly necessary to allude to the paralysis following dislocations, for of course the surgical proceeding, which is indicated at first, is the reduction of the luxated bones, and this should be done as early as possible.

In the management of paralysis, which, Desplats<sup>3</sup> says, may be due to pressure made by osseous enlargements, iodide of iron and other proper remedies, with cod-liver oil, are to be employed. If there be neuritis, it should be met with counter-irritation, emollient applications, or leeches.

General supporting treatment may be necessary if there be a depraved condition of the system.

The three valuable local forms of treatment are: 1. Electricity; 2. Strychnia, internally or hypodermically; 3. Massage.

The first agent may be used as early as possible. If one current will not produce contractions, we may use the other; and, if complete severance of the nerve has taken place, it may be necessary to employ galvanism. Faradism is especially valuable should there be anæsthesia, and may be applied to the cutaneous surface. The galvanic current may also be used at the same time, so that one electrode shall be applied to the spine, and the other to the extremity. The individual muscles are to be subjected to daily galvanic stimulation.

The production of pain is unnecessary, and I may repeat the clinical rule so tersely applied by H. C. Wood:<sup>4</sup> “Always select the current

<sup>1</sup> Centralblatt für Med. Wiss., July 13, 1872.

<sup>2</sup> Comptes Rendu, 1872, No. 15.

<sup>3</sup> Dés Paralyes Périphériques, Paris, 1876, p. 45. <sup>4</sup> Phila. Med. Times, Feb. 20, 1875.

which produces the most muscular contractions, with the least amount of pain." Pain and over-fatigue, which follow the use of a strong current, are very apt to thwart any probable success. The application should last not more than ten or fifteen minutes every day.

An excellent method of treatment is to place the paralyzed limb in a vessel of warm salt water, and to introduce therein two metallic plates connected with a faradic machine. If there be neuritis, induced electricity does great harm and should not be used.

I have repeatedly witnessed the beneficial results which followed the use of hypodermic injections of strychnia. An injection of  $\frac{1}{30}$  of a grain may be thrown under the skin over the paralyzed muscles. This may be repeated daily; and I have sometimes seen its good effects when electricity was without avail.

The use of "massage" should be employed in conjunction with the other treatment, and the muscles should be separately kneaded and rubbed for an half hour each day. This auxiliary treatment is of immense value when there is suspected rheumatic exudation.

I have often employed apparatus by which the paralyzed limb could be subjected to warmth, and for this purpose have used a heated drain-pipe lined with cotton-wool, such as has been spoken of on another page. Into this the patient was directed to place his arm and allow it to remain for an hour or so each day. The paralyzed limb may be wrapped in cotton and oil silk, or India-rubber tissue.

The union of divided ends has been resorted to by Tillaux,<sup>1</sup> Nélaton, and others, and with a great deal of success. In Tillaux's case the median nerve was united by sutures, and within a day or two the patient was able to move his thumb, and there was some return of sensation.

Mitchell<sup>2</sup> employs the following method: He carries a needle, threaded with one or two threads, through the loose tissue which is related to the nerve-sheath. The loops are drawn with care, so that the ends are approximated. Hot and cold douches and electricity are subsequently used.

In some cases we may use Van Bibber's apparatus.

Van Bibber presented the following case to the Maryland Medico-Chirurgical Society which illustrated the beneficial results of treatment of this kind:—

"A youth, æt. 16, about three years ago sustained a fracture of the right radius, which resulted in paralysis and atrophy of the extensor group of muscles. He first came under my observation about three months ago, when I found the following condition of the arm: radius curved; hand flexed, and the flexors acting inordinately; complete atrophy of the extensor muscles, it being impossible for him to move his hand; no response of the muscles to electricity; and the skin tightly bound over the radius. The treatment has consisted in rubbing and pinching the af-

<sup>1</sup> Quoted by Weir Mitchell, *Dis. and Inj. of Nerves*, p. 238.

<sup>2</sup> *Ibid.*, p. 243.



fectured muscles, the application of electricity, and the use of the artificial muscle, which is nothing more than an elastic tubing fixed to the back of the arm. The results of treatment have been very satisfactory; the lost muscles have been restored, the skin has regained its former tone and elasticity, and the motion is fast returning."

I may in conclusion present a case which was reported by Bernhardt, in which electricity was used.

"L.,<sup>1</sup> 43 years old; dislocated his left humerus by falling on his left shoulder. He had pain in the shoulder, and found it impossible to use his arm, and that felt cold. The dislocation was found to be subcoracoidal, and after eight days it was reduced. The pain ceased, but the paralysis continued. In the palm of the hand there was, after three weeks, considerable scaling of the epidermis. Pressure on the shoulder was not painful, but a strong grasp of the triceps and of the muscles of the forearm was unpleasant. Occasionally there was a sense of formication from the middle of the arm down the extensor side of the forearm to the end of the fingers. The left arm could be raised in a straight line forward about half a foot, but could not be carried backward nor across the breast. The forearm could not be bent on the arm; only the supinator longus was rendered tense. Extension was impossible; supination was slight. The hand could be raised somewhat. Abduction and adduction of the hand, flexion and extension of the fingers, were impossible. The prick of a needle was felt to the upper border of the lower third of the arm on both sides equally. In the lower third of the left arm, in the elbow-joint, and the upper part of the forearm, the skin is more sensitive on the right than the left. In the rest of the forearm, in the hand and fingers, the sensation is a little less on the left than right, but nearly equal. The muscles of the arm and forearm, of the hand and finger, as well as the deltoid, showed only the slightest reaction to the induction current. Likewise the use of a very strong galvanic current either to nerve or muscle, by opening or closing, failed to produce contraction.

"From the 5th of January, every other day, the patient was treated with a strong galvanic current, the anode and the cathode being placed on the paralyzed muscles. After four weeks he could raise the arm forty degrees, also some distance backward, so as to touch the right shoulder with the left hand. Also, he could bend the forearm on the arm, and had some motion in the hand and fingers. After eight weeks more, motion was nearly restored.

### DIPHTherITIC PARALYSIS.

Diphtheritic paralysis may either take place as a feature of the diphtheritic attack, or it may appear during convalescence, or even several weeks after recovery. The paralysis is generally bilateral, and does not last any great length of time if the throat is alone affected, and rarely exceeds ten or fifteen days in duration. Should the loss of power begin at the same time as the acute disease, the progress of the case is much more apt to be favorable, and the paralysis disappears in a shorter space of time than if it occurs at a period subsequent to the disease.

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<sup>1</sup> Berliner Klinische Wochenschrift, No. 5, 1871.

Lanné states that a marked and sudden increase of temperature during the diphtheritic attack or convalescence is indicative of paralysis.

The paralysis may be simply motorial, or there may be a corresponding loss of sensation which is variable in extent.

The muscles of the throat are usually involved, so that regurgitation of fluids takes place through the nose, or there may be certain phenomena which are so well marked in bulbar paralysis, in which the lesion is one of a destructive character. When the limbs are paralyzed, there may be, according to Brenner, movements of a choreic character which depend upon the irregularity of the paralysis, the antagonism of certain groups of muscles being abolished. The organs of special sense are not unusually involved. There may be paralysis of the muscles of accommodation,<sup>1</sup> neuro-retinitis, and sometimes ptosis. Deafness is not rare, and in one of my own cases there had been tinnitus immediately preceding the deafness.

The following case is of a very interesting nature, from the fact that it is reported by the patient himself, who is a medical man.<sup>2</sup>

"In October, 1875, being twenty six years of age and in good health after two months' constant exposure to diphtheria, I was inoculated from a child two years old, who, on examination, coughed portions of the membrane into my face. Six days after this exposure I was seized with a chill, followed the next day (October 28th) by the appearance of a diphtheritic deposit on one tonsil. The deposit was limited to the tonsils and back part of the pharynx, and in nine days disappeared. Exhaustion and great gastric irritability retarded convalescence. Four weeks passed before I was able to sit up. Two weeks after convalescence was declared, a sharp, lacerating pain in the left axilla was noticed, recurring two or three times at short intervals. In a few days, after seeing visitors or talking a little, severe and constant pain in the elbow-joints occurred, which soon extended to the muscles of the arm and chest. After resting, these pains diminished or disappeared, and in a week entirely ceased. On attempting to rise, my limbs seemed surprisingly weak, but at the expiration of the sixth week a short walk was possible. After a brief period of improvement my legs began to grow uncertain and weak, and by December 10th I could take but a few steps. At this time a partial loss of sensation came on, beginning in the feet and gradually progressing to the trunk, together with a feeling of coldness in the feet, which, however, were not cold to the touch. This numbness increased faster than the loss of motion. Soon after its appearance in the lower extremities the ends of the fingers lost their sense of touch, the loss of power also extending in a week to the elbows, and at no time greatly affecting the arm. Loss of motion in the fingers and forearm accompanied it, and increased for some weeks. The mouth, tongue, and portions of the face lost their sensitiveness at the same time and to the same degree. In a few days my voice grew thick, and was soon like that caused by cleft palate. The soft palate and uvula hung loosely in the mouth, and on attempting to swallow fluids they were regurgitated through the nares.

<sup>1</sup> See cases reported by Hutchinson, *Lancet*, Jan, 7, 1871.

<sup>2</sup> Dr. A. F. Ræd, *Boston Medical and Surgical Journal*, July 13, 1876.

Dimness of vision for a short time prevented reading. In three weeks my voice, then at times unintelligible, grew suddenly better, and in four or five days was restored. The difficulty in swallowing also soon disappeared. The loss of motion and sensation in both arms and legs increased. In walking I seemed to be on velvet; there was a sensation of coldness in my feet, and at first the circulation was retarded. The general loss of power was progressive until February 1st. It was then impossible for me to stand alone even when lifted up, to raise myself an inch from the chair by my arm, to bring my thumb and forefinger together, or to exercise my strength in any part. The toes hung lifeless, and no reflex action was produced on tickling the sole of the foot. The urine was voided with difficulty, and the power of erection was gone. The interosseous muscles were wholly paralyzed, though still reacting to the faradic current. The fingers were drawn up when the hand was at rest, but only by great effort could be straightened out again. The muscles of the arms were much weakened, but with those of the thigh retained more power than the rest. They were also the last to lose and the first to gain motion. All these muscles were more or less responsive to the faradic current, the gastrocnemius least of all. During the weeks previous and at this date my appetite was excellent, and my food well digested. From this time an improvement as general as the invasion was noticed. In one week I could lift my body in the chair an inch or two, and when standing felt more secure. In two weeks I could raise myself up from the chair mainly by my arms, and undressed without aid. At the end of three weeks I could walk about the room aided by a cane, and wrote legibly. The difficulty in voiding the urine and loss of power of erection had by this time gone. In four weeks I walked out for a short distance, and in two weeks more all paralysis had disappeared, leaving some neuralgic pains in the knees and feet, which lasted but a short time. On April 1st I walked several miles without great fatigue. Atmospheric changes made no change in my strength. Insomnia was the greatest annoyance suffered while confined to the house. Three or four hours' sleep was all that could be obtained. The loss of sleep did not, however, leave me unrefreshed.

"Treatment: From January 12th faradism to the muscles every day until February 15th, afterwards three times a week for three weeks. Tincture of *nux vomica* and tincture of phosphoric ether were given for ten days. The stomach rejecting these, one-thirtieth of a grain of strychnine was substituted, which was increased to one-fifteenth three times daily for six weeks. A pint of ale daily for two months. Friction and kneading of muscles every morning for one hour."

**Causes.—Morbid Anatomy and Pathology.**—Dowse<sup>1</sup> quotes Balthazar Foster, who has stated that "he has never known paralysis to follow the non-febrile form of diphtheria." Dowse thinks that the violence of diphtheria has little to do with the development of the paralysis, and says that he has seen cases following modified attacks.

My own experience leads me to disagree with him. I have seen six

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<sup>1</sup> See case reported by Dr. A. W. Foot, Dublin Quarterly Journal, Sept. 1872, p. 176, of "Locomotor Ataxia subsequent to Diphtheria." This was evidently the ataxic form of Brenner.



cases of diphtheritic paralysis, and these were among the most violent cases.

Labadie Lagrave, Andral, and others have called attention to the blood-changes in this disease, viz., diminished fibrine and an increased number of white corpuscles. Sauné has found that the red corpuscles are destroyed, and that there is a great increase in the amount of *débris* with albuminous urine. The paralysis takes place, however, in a later stage, but Dowse has shown that the albumen in the urine reappears with the paralysis, and that it again diminishes in quantity as recovery takes place; hence we may infer that a connection exists between the blood condition and the paralysis. I am inclined to think that the paralysis of the palate and muscles of the pharynx are the results of pressure made by the diphtheritic membrane.

**Diagnosis.**—Diphtheritic paralysis need not be mistaken for any other affection, though occasionally, in its ataxic form, it is confounded with posterior spinal sclerosis. Its transitory nature should render such an error as this impossible. For the same reason it should not be confused with organic paralysis.

**Prognosis.**—I have never heard of a fatal case, that is, a death which was a result of paralysis occurring during convalescence from diphtheria. When paralysis takes place before the violence of the disease has been spent, death may take place from the acute disease. The duration of the paralysis is from eight or ten days to many months.

**Treatment.**—Nutritious food, massage, strychnia, and iron, quinine, and stimulants with faradization, are the indications. The plan pursued in Dr. Reed's case will serve as a model for others to go by.

## CHAPTER XIX.

## DISEASES OF THE PERIPHERAL NERVES (CONCLUDED).

## LEAD POISONING.

**Synonyms.**—Colica pictonum ; Plumbism.

The toxic effects of lead, whether taken internally or absorbed by the skin, are extremely varied and interesting. Disorders of motility and sensation are produced which, though rarely alarming, are most distressing conditions.

**Symptoms.**—Among the early symptoms of lead poisoning may be mentioned the abdominal pain which has received the name of *colica pictonum*, and which Romberg<sup>1</sup> considers a species of neuralgia of the mesenteric plexuses. Tanquerel<sup>2</sup> has graphically sketched the appearance and development of this symptom. At first there is constipation which lasts for some weeks, and sometimes follows a slight diarrhœa, while after a short time a sense of epigastric oppression is experienced, with nausea and eructations, and gnawing twisting pains which occupy the umbilical region. These pains are much worse at night, and rarely shift their position. Pressure relieves them to some extent, as it does in simple colic.

During the paroxysms there is great muscular rigidity, and the abdominal muscles seem to be rigid. The skin is cool, and perhaps bathed in sweat, and the pulse is full and bounding, and quite hard. The constipation continues, and the feces that are occasionally voided are scybulous and of a whitish-gray color. The urine is of high specific gravity, is quite light in color, and voided in considerable amounts.

The complexion of the individual is sallow, and the skin rough ; and, if his lips be separated, the peculiar bluish line at that part of the gums which is in contact with the teeth will be seen. This line is a quite constant symptom ; it is perhaps one of the most valuable diagnostic marks. The remaining part of the gums is quite spongy and dark.

There may be in conjunction with lead colic a very well-marked cutaneous anæsthesia or hyperæsthesia, but the latter is more common. The skin is exquisitely sensitive in parts, such as the scalp, the groin, the bend of the elbow, and other like regions. Pressure seems to relieve this tenderness, but light irritation aggravates it markedly.

A form of tremor which is apt to be confused with those of a sclerotic nature has been found as a rare symptom. Brockman observed it among

<sup>1</sup> Op. cit. vol. ii. p. 132.

<sup>2</sup> Traité des Maladies de Plomb. ou Saturnines, 1830.

workers in the lead mines of the Hartz Mountains. It may be local or general, and in the first form the hands are affected. The lips may be agitated, and the levator anguli oris is often involved, so that the corner of the mouth is drawn up. In the other form the head, trunk, and arms are all in a state of tremor, the head being bowed on the chest, and the legs unsteady. In this latter form there is usually a profound toxic condition.

By far the most important symptom, and one which may or may not be preceded by lead colic, is the form of local paralysis known as "lead palsy" or "lead paresis." The onset of the malady is usually gradual, the patient being unable at first to extend the fingers. There is nearly always some numbness of the hand, and rarely tremor. It is not often that the paralysis becomes general, but the extensors of the forearms are, as a rule, involved. In this condition the hands hang helplessly, and an appearance results which has been called "drop wrist." There is generally some paralysis of the flexors, but this is almost inappreciable. Other muscles, notably those of the shoulder, are affected if the lead saturation be profound, and, as a consequence, the patient may be unable to raise his arm. I have never seen a case in which the lower extremities were involved.

Electric sensibility and contractility are much reduced, and there is marked anæsthesia in most of the cases. Faradism rarely provokes muscular contractions, and in old cases even the galvanic current fails to call forth the slightest response.

Atrophy is a result of the paralysis, and the interosseous spaces of the forearm are sometimes very plainly marked, the loss of substance being quite decided.

The colic generally subsides with the appearance of the paralysis, and according to Romberg<sup>1</sup> the two conditions rarely co-exist. In the cases recorded by various observers the muscles of *both* extremities of one kind were affected in the great majority of instances, and from my own experience I consider unilateral lead paralysis to be an anomalous condition, but impaired function not equal.

Occasionally a cerebral condition results from lead poisoning, and generally follows the colic. This is characterized by vertigo and headache, general malaise, and tremor of the hands which is aggravated by voluntary action. A more serious state is sometimes produced, however, which is symptomatized by delirium, convulsions, and stupor.

The duration of lead paralysis, or the other conditions I have noticed, is of course governed by the existence of the cause and the exposure of the patient. Most of the toxic lead states disappear, however, in a very short time, provided the patient protects himself by leaving his injurious occupation, and the proper remedies be administered.

The following may be cited as a well-marked case of lead poisoning:—

Jas. McK., æt. 55, N. Y. City, painter. Has followed his trade 35 years, engaged mostly on "inside work," "flatting." Never had any

<sup>1</sup> Op. cit., vol. ii. p. 126.



trouble till two years ago, when he noticed pains in his limbs, back, and suboccipital region; not much colic, but some nausea; loss of appetite; not constipated. While actually engaged in work he became dizzy, and "a blur came across his eyes." Last acute attack was obliged to leave work suddenly on account of severe backache. He then noticed a loss of power in right hand.<sup>1</sup> He consulted me in July, 1877, presenting well-marked "wrist drop," so that he was unable to extend his hand. He complained of formication of soles of feet, insomnia, and pains in shoulders, knee-joints, and about heart. Well-marked blue line and very dirty gums. The necks of the teeth are carious and black, and he has lost several of them during the past few years.

Loss of sensation of cutaneous surface.

*Hand*.—Atrophy of adductor of thumb, so that quite a hollow exists.

*Forearm*.—Complete loss of electro-muscular contractility in common extensor of right forearm; slight power under electrical stimulus of extensor of thumb and little finger. Flexors slightly impaired, but contractility scarcely lost.

*Arm*.—Muscles all contract well. Patient cannot take off his coat or underclothing, or cannot button his clothes.

*Treatment*.—Electricity and potass. iodid. with strychnine.

**Causes.**—The majority of cases of lead poisoning arise from the inspiration of finely divided particles of lead, and not from the manipulation of pieces of the metal; consequently, painters, smelters, white-lead makers, and miners are more often victims than any other classes of individuals. There seems to be an idea that printers are especially subject to lead diseases; and at the request of the Board of Health of the city of New York I made an extensive examination of the printing-offices for the purpose of testing the question. I interviewed nearly 1500 men, women, and children, and found not a single case of paralysis. Among the grinders of type (those who smooth the sides and ends of the type against large rough stones), I found that the persistent use of the muscles of the thumb and forefinger, in one case, resulted in a condition resembling progressive muscular atrophy. In the lead pipe and shot manufactories my experience was the same.

The painters, however, seem to be most frequently poisoned. An operation known as "flatting," in which the painter closes all the doors and windows of a room, and applies thin paint, is attended with great danger. The turpentine evaporates rapidly, and carries with it minute particles of lead which the workman must inhale.

Dr. Richardson,<sup>2</sup> in a thesis which embodies a large amount of valuable research, thus describes the manner of preparing white lead, and the danger which attends its manufacture.

"The metal first comes in contact with the skin of the men in being carried by hand from the cars to the melting-room. Here many tons are melted at once and cast into thin, circular, perforated plates called buck-

<sup>1</sup> Can only force dynamometer index to 4 with right hand; left, 15.

<sup>2</sup> Graduation Thesis, Harvard Medical School—Boston Med. and Surg. Journ., Oct. 4, 1877.

les, of such shape as to expose as much surface as possible for the weight. The temperature is very high. Bathed in perspiration the men stand for hours inhaling the minute particles of the oxide of lead which escape from the cooling buckles and fill the air. Their thirst in this part of the process is insatiable, and enormous quantities of ice-water are swallowed, whereby the dust, which adheres to the tongue and lips, is washed directly into the stomach.

Having been carried to a neighboring shed, the buckles are placed over pyroligneous acid in earthen pots of about four quarts capacity. Many thousands of these pots are packed together in the refuse of stables or the exhausted bark from tanneries, and are exposed to the moderate heat which is spontaneously generated about them. The wood vinegar is volatilized and rises through the buckles, changing by some obscure chemical reaction the blue metallic lead into the white carbonate. After an exposure of this sort, lasting from six weeks to three months, the pots are unpacked and the whitened lead removed. Here for hours men breathe the vapors rising from the heated bark, loaded with poisonous particles of the now dusty metal. In English mills this part of the process is done by women, with most disastrous effects upon the health. To separate the blue from the white lead the buckles are placed in a revolving cylinder of wire-cloth, through which the carbonate, more or less pulverized, falls. The blue portion remains in the cylinder and is melted again. To be in this room without protection is suicidal, for the air is filled with visible clouds of dust. The utmost care must be taken. The mouth and nostrils are covered by a moist sponge to catch the floating particles. The skin and clothes quickly become white with lead. The semi-powdered metal, having been shovelled into barrels and rolled into another division of the works, is mixed with water and finely ground. When it fills the water as a milky precipitate, the whole is drawn off and dried on long tables at a temperature of  $140^{\circ}$  F. Formerly the grinding was done without water, and the lead sickness was much more common than now. The drying-room is the most poisonous one in modern mills. It combines the effects of the dust which fills the air with those of a heated atmosphere. Here, as in the melting-room, the skin is kept in the best state for absorption. A terrible thirst makes the men swallow large quantities of cold water with the lead which accumulates on their lips and tongues, while at every breath fine dust is drawn into the lungs.

The general appearance of the men is not good. The faces are sallow and more or less worn. The sclerotic coat is yellowish. Their motions are far from energetic, and in some cases eccentric and unsteady. One would say immediately, I think, that the general appearance is much below that of the average workman.

1. The first man examined has worked in all parts of the mill for thirteen years. His only trouble is rheumatism. The gums show a distinct blue line along the border.

2. After seven years in the corroding rooms has no symptoms excepting the blue line.

3. After grinding lead with oil has only the blue line.
4. After working in all parts of the mill for six months has had violent colic and great constipation. Blue line marked.
5. Reports only blue line after four years' work.
6. The machinist, after repairing in the drying-room a few hours a day for ten days, was affected with colic and constipation. Has great habitual constipation. Blue line very marked.
7. After seven years only blue line.
8. After twelve years has only blue line and fungous bleeding gums, with occasional colic and obstinate constipation.
9. After six years in corroding-room has only blue line.
10. Has worked in all parts of the mill for fifteen years without showing a trace of blue line or any other symptoms whatever. Very neat.
11. After three years only blue line.
12. After four years, nothing.
13. Blue line, rheumatic pains, and fainting fits. This was a remarkably neat man.
14. After four years no trace of poisoning.
15. After four years entirely used up. Had to leave all work.
16. After one year's work completely crippled, having paralysis of the extensors, aphonia, and general debility.
17. The carpenter, after repairing ten days in the drying-room, had severe colic, obstinate constipation, and persistent blue line.
- 18-75. Of the rest of the seventy-five men whom I examined all had a distinct blue line about the gums, and, with one or two exceptions, habitual constipation. There was nothing further than this to suggest the presence of lead.

In addition to the above cases, three of the former employés had suffered with difficulty in speaking, three with amaurosis, several with cerebral troubles, and many with paralysis. The superintendent has observed that the most frequent complaint has been of swollen joints and aching bones. In the numerous cases of paralysis which he has seen during many years' service at these works, he has noticed that the wrists have become much swollen before paralysis of the extensors. A curious tradition exists among them that they cannot drink alcoholic liquors and keep up with their work, like laboring men in other manufactories. Several cases were told me of men who quickly succumbed to the influence of the lead after beginning the use of strong stimulants."

Lead is often taken into the stomach without the knowledge of the individual, and lead pipes are a prolific source of the contamination of water. I have seen three cases in the same family caused by tea which had been made from a specimen containing particles of sheet lead which had lined the box. The last two or three pounds were impregnated with these impurities, which had settled to the bottom of the chest. It was the custom to make tea and from time to time to add fresh leaves and pour on hot water, so that there was constantly a quantity of lead sub-



jected to the action of the fluid. Upon analysis, quite an amount of lead was found.

Cases arising from the use of cosmetics and hair-dyes are two common to need anything more than bare mention.

**Morbid Anatomy and Pathology.**—Andral and Tanquerel<sup>1</sup> were unable to discover any pathognomonic condition of the intestines in lead colic; but the latter authority found lead deposits in the intestines, muscles, and nervous substances. In a case of lead paralysis reported by Gombault,<sup>2</sup> there was found to be no change in the cord, and the only morbid appearances anywhere else were in the nerves, the medullary substance having undergone a granular alteration. No other appearances which might clear up the pathology of the affection have been seen.

Remak<sup>3</sup> is of the opinion that lead palsy is a central disease, and he presents several cases to show its likeness to infantile paralysis. The same electrical reaction of the muscles in these two affections, and the fact that groups of muscles are affected which act together, not necessarily being those supplied by the same nerve, leads him to think that the paralysis is of central origin. The blue line of the gums, which indicates plumbic saturation, was first described by Burton in 1840. By Tanquerel it is supposed to be produced by the decomposition of food about the teeth, the sulphuretted hydrogen uniting with the lead. It occurs in people who brush their teeth as well, however, as in those of careless and untidy habits. Dr. Richardson<sup>4</sup> tried the following experiment:—

“A strong, healthy cat was fed for a week upon milk, to which had been added a small portion of a solution of plumbic acetate. At the end of a week the animal was killed, after having shown symptoms of severe constitutional disturbance. The lower jaw was excised, and the gums found perfectly clean. The upper jaw was also clean. The lower jaw was placed in water, through which a stream of sulphuretted hydrogen was passed for several hours. At the end of that time a perfectly distinct and unmistakable blue line was found throughout the juncture of the gum with the teeth. The stomach and intestines of the animal showed nothing remarkable. The presence of the blue line seems, therefore, to depend on a certain amount of putrefaction about the teeth.”

The elimination of lead is usually rapid when the proper remedies are administered to convert it into a form for excretion. If nature is left to herself, the process is more slow. Potain considers that it is eliminated only very slowly by the sweat-glands, and not by the kidneys or salivary glands, but I am disposed to consider that elimination *does* take place by the kidneys.

<sup>1</sup> Tanquerel, p. 326.

<sup>2</sup> Archives Générales, 1873.

<sup>3</sup> Archiv für Psychiatrie und Nervenkrankheiten, vi. p. 1.

<sup>4</sup> Op. cit.

**Diagnosis.**—In nearly all cases of lead poisoning, it is usually possible to detect the cachexia, which is so well expressed by the different signs I have enumerated. If our suspicions are not verified by appearances in an acute case, we may test the patient's urine. A few drops of a solution of potassic sulphide will usually precipitate any lead that may be present in the form of a black sulphide.

The paralysis may be sometimes confounded with other forms, but when it is remembered that the extensors are prominently affected; and that there are lead symptoms at some time or other, it is not possible to be mis'taken.

Dr. Wharton Sinkler,<sup>1</sup> in an admirable paper, calls attention to the resemblance between "wrist drop" due to lead poisoning, and paralysis of the extensors from injury of the musculo-spiral nerve. He has found paralysis of the flexors of the forearm after injury of the nerve, and he is inclined to think that in the beginning there is never paralysis of the flexors in lead palsy. In lead paralysis the supinators escape.

**Prognosis.**—With the disappearance of the cause, we may expect in most cases a rapid subsidence of symptoms. It is true the paralysis often lasts for some time, but even this ultimately disappears. Deaths by lead poisoning are rare, and I suppose when they occur are due to an affection of the brain, to which I have alluded. The mortality from lead poisoning in New York City from 1852 to 1873 was 288. 48 died in 1852; and, strange to say, but four in 1872.<sup>2</sup>

**Treatment.**—If we have correctly diagnosed the condition, our objects must be: 1. To relieve pain; 2. To favor elimination of the lead; 3. To guard our patient against being continually affected; 4. To restore the paralyzed limbs.

1. No better remedy is possessed than iodide of potassium, which forms an iodide of lead which is an innocuous salt. This drug must be given in moderate doses,<sup>3</sup> and its elimination hastened by mild purgatives. It will be found that, if the patient is obliged to continue at his work, small doses taken daily, or acidulated drinks, will, in some measure, prevent the absorption of lead. If there be colic, the hypodermic use of morphine will give great relief.

It has been found that those workmen who drink a great deal of milk seem to escape the danger of lead-poisoning. In France the workmen in the lead-works are obliged to drink milk, and it is found to be an excellent prophylactic. Richardson's case (*loc. cit.*) did not suffer so long as he kept his cows; but when he parted with these animals, and stopped drinking milk, the most decided symptoms of plumbism manifested themselves.

As to the employment of electricity, it is well to use the faradic current if possible; but in some cases this produces no contractions. In

<sup>1</sup> Am. Psych. Journal, Nov. 1875, p. 31.

<sup>2</sup> Report of the Board of Health, 1872.

<sup>3</sup> Very large doses seem to increase the symptoms.

such an event we may begin with the slowly intermitted galvanic current; and, after a while, it will be found, as in some other paralyses, that the faradic will cause muscular response, particularly if the arm be so supported that the muscles shall be relaxed. Dr. H. C. Wood,<sup>1</sup> of Philadelphia, has noticed the fact that voluntary power may return to a great degree without a corresponding return of electric contractility.

I have before alluded to an instrument devised by Dr. J. Van Bibber,<sup>2</sup> and it is well to apply this so that the muscles may be entirely supported.

In conclusion, I may present the records of a representative case of lead palsy. The patient was under the care of Dr. Cross, through whose kindness I had the opportunity of seeing him:—

M. C.,<sup>3</sup> aged 32 years, single, born in Ireland, a painter by occupation. He has been moderately temperate in his habits, and has always enjoyed good health until 1863, when he was suddenly seized with a severe attack of colic, which was preceded by great constipation of the bowels and loss of appetite. There soon succeeded nausea and vomiting of bile, accom-

<sup>1</sup> Phila. Med. Times, Feb. 20, 1875.

<sup>2</sup> "After many attempts to secure this advantage by means of strips of plaster, it was determined to try the India-rubber muscle as used by Dr. Lewis A. Sayre in orthopedic surgery. The great difficulty in the use of such an appliance was to effect its application without causing injurious pressure upon the circulation of the arm and hand. I am not aware that these elastic tubes have been used before to correct this deformity, or attached by a method so simple and so free from pressure as that which I shall now describe. Two bands of inelastic webbing, pierced by eyelets at certain points, and each having a convenient buckle, serve as points of attachment. The one for the hand, about three-quarters of an inch wide, so made, that the free end placed upon the palm pointing toward the thenar eminence, and the eyelet-hole resting on the ball of little finger, the band folded once around that finger and passed over dorsum of the hand, the buckle would come in a convenient place upon the palmar surface. The band for the arm about one inch in width, so arranged that the eyelet being placed upon a line a little above the external condyle, the buckle would rest upon the internal surface of the arm.

As seen by the illustration, two transverse strips of plaster are adjusted to the arm so as to form an angle just below the eyelet, and thus relieve the band, which should be buckled loosely, from all injurious traction. The fold around the little finger, and the muscle resting upon the webbing on the dorsum of the hand, enable us to buckle the band loose enough to insure perfect abduction of all the fingers. Finally, a piece of India-rubber tubing of correct length and medium elasticity, with one of Dr. Sayre's metallic hooks attached at each end, constitutes the entire apparatus.

Looking upon this artificial muscle as performing to some extent the duty of those paralyzed, I can probably best describe its application by saying, in anatomical language, that it arises from a point a little above the external condyle, and passing downward on the extensor surface of forearm, under the cuff, which we might call the annular ligament, forward over dorsal aspect of the hand, passing between the index and second fingers, which serve as a trochlea or pulley, then transversely across the palmar surface of the hand, and is inserted at a point about the articulation of the fifth metacarpal bone with its first phalange."—*N. Y. Medical Journal*, May, 1874.

<sup>3</sup> Reported in the *Psychological Journal*, Jan. 1871, by Dr. Cross.



panied by an acute lancinating pain in the epigastric region, which was so severe that the patient was obliged to lie flat on the floor and press his abdomen strongly against that surface, in order to obtain temporary relief. These symptoms continued off and on for a period of about two weeks, gradually diminishing in severity, however, especially after an evacuation from the rectum, which was only obtained with the greatest difficulty. His right leg at this time became œdematous. In the course of two months he resumed his usual avocation, that of a painter, but was not aware at this time that his sickness had been caused by the action of lead. During the year 1867 his bowels again became very costive; and his stools, which consisted of only a few lumps of dry, hardened feces, were attended with much straining.

Soon there followed a second attack much more severe than the first, which was characterized by nearly similar symptoms, only there was superadded great tenderness over the kidneys, which were so sensitive that the least pressure caused him the most intense agony. The urine was very scanty and high-colored, and there was a well-marked blue discoloration of the gums. In a few months, having somewhat recovered, he went to work again at his former occupation, which he pursued uninterruptedly until the 25th of December, 1869, when, after having passed a very uncomfortable day, his former symptoms returned with increased violence, while the paroxysms of the colic came on at much shorter intervals than they had done in the preceding seizures; in fact, instead of intermissions as formerly, there were only remissions of the intestinal spasm. For the first time he had pains in the feet and the inside of the thighs. The urine was more scanty and higher colored, and the bowels more constipated than before.

In three weeks he again began to work, and had no more trouble, except constipation of the bowels and weakness in both his upper and lower extremities, until July, 1870, when he lost his appetite, and felt very weary and exhausted after any small amount of exertion. He was very restless and could not sleep at nights, and this inability to sleep was a sequela of all the other seizures. Now came great tremor of the right hand and arm, which was soon followed by tremor in the left.

In August, 1870, he had his fourth and last attack, which was the most severe of all, and lasted about two weeks. This time he vomited blood, had acute pains in the soles of his feet, and cramps in the right hand. On recovering from the effects of the colic he found that he was unable to use his arm or hand at all, and that he had lost power in his legs also.

Soon after this he was admitted to the Charity Hospital, where he remained for a fortnight, and during his residence in that institution he became delirious, and continued so for about eighteen hours. He came to the out-door department of the New York State Hospital for Diseases of the Nervous System, September 12, 1870, when his condition was as follows: There was the characteristic drooping of both wrists, which was very extreme in degree. The paralysis of the supinator and extensor muscles of both upper extremities was exceedingly well marked; the flexors were also involved, only to a much more limited extent. The paralysis was more considerable in the right forearm and hand than in the left. There was much atrophy of all the muscles of these parts, and this was very conspicuous in the abductors and adductors of the thumbs. The patient was so very weak in his lower extremities that he was unable to arise from the sitting posture without assistance, and as he walked he

tottered at every step. Yet he did not drag the toe of either foot, nor swing his legs, as do those suffering from hemiplegia. The blue line was very plainly seen around the edge of the gums of the upper and lower jaws. On testing the amount of muscular power in the right hand by means of the dynamometer, he was able to turn the indicator only 10 degrees, while with the left he could accomplish somewhat more. The tactile sensibility and the sensibility to the electric current and to pain were very greatly diminished. The temperature was also diminished; muscular contractility was so much impaired that a powerful induced current had not the slightest effect in causing contractions, and, even when the primary galvanic current (sixty cells and very strong) was used, the muscles responded very feebly, if we except, perhaps, the flexors, so almost completely had their irritability been destroyed. The bowels were regular, the urine was normal, and, although no chemical analysis for lead was made, undoubtedly it would have been found. "The appearance of the patient was anæmic, cachectic, and depressed; the breath was very offensive; the retinæ were anæmic; the lungs were healthy, and so was the heart, excepting an inorganic murmur at its base."

The treatment in this case has consisted of the internal administration of the iodide of potassium, commencing with ten-grain doses three times a day, and the daily application of the primary galvanic current to the paralyzed muscles, with a hypodermic injection of the thirty-second of a grain of the sulphate of strychnia every day.

*September 17.* The iodide was increased to fifteen grains three times a day.

*24th.* Slight fibrillary contractions in the right arm were produced to-day for the first time by means of the faradic current.

*October 1.* The iodide of potassium was increased to twenty grains three times a day.

*5th.* The induced current had just commenced to cause slight contractions in the left forearm.

*November 15.* Faradization of the left forearm produced good contractions in the extensor carpi radialis and ulnaris muscles. The blue line having disappeared, the iodide of potassium was discontinued, and a tonic substituted.

*23d.* The muscles of both arms respond feebly to the induced current, yet by means of it the hands can now be extended nearly on a level with the forearms. The right has improved the most. Sensibility to touch and to electricity has much improved. His bowels are regular, he sleeps well, and his appetite is good. The power in both hands is much increased, and he is able to work every day.

*January 1, 1871.* The patient has almost entirely recovered.

## FUNCTIONAL SPASM.

Under this head I propose to include the various forms of hyperkinesis which depend upon irritability of the nervous centres, and which have been specially considered, as *Tetany*, spasm with voluntary movements, *Reflex Spasm*, *Torticollis*, *Professional Cramp*, etc.

These are generally due to some peripheral cause, or may result from overtraining of the automatic sense, or in certain conditions arise in a manner which is at present not clearly understood.

## I. TETANY.

A light form of attack arising generally from diarrhœa, cold and constipation, and sometimes making its appearance during lactation. There is usually some formication of the palms or soles, and an awkwardness in the movements of the hands and feet, which is afterwards followed by a firm tonic contraction of the muscles of either of these parts. The flexors are usually contracted, so that the hand is curved, or all the fingers closed. A more decided contraction may flex the forearm on the arm. The foot may be also affected, a condition of talipes resulting, or the back part of the leg may be brought in apposition to the thigh. In marked forms the upper and lower extremities are affected together, though there is no rule governing this, and the spasm may be bilateral or unilateral. The attack rarely lasts beyond an hour or two, and in the majority of instances relaxation may take place in from five to ten minutes. The spasms may come on from time to time, being separated by greater or less intervals. They are entirely uncontrolled by the will, and the patient cannot open his fingers when they are thus contracted. In more severe forms the muscles of the trunk or face become involved. Contraction of the ocular muscles, laryngeal spasm, trismus, or vesical spasm are examples of more violent action. The spasms seem to be produced when pressure is made upon a nerve-trunk or muscular belly, and there is loss of tactile sensibility associated with neuralgic pain in the main nerve trunk of the convulsed limb.

Tetany differs from true tetanus from the fact that the spasms affect all the limbs, that they are intermittent in character, and that there are intervals of relaxation. *Petit-mal* sometimes resembles this condition, but there is always some loss of consciousness.

## II. FUNCTIONAL SPASM WITH VOLUNTARY MOVEMENTS.

Mitchell<sup>1</sup> reports some cases of functional spasm, which somewhat resembles the so-called tetany. The spasm appeared during the exercise of a voluntary act; they occur with the act of laughing, chewing, and talking, and evidently depend upon functional derangement of muscles innervated by the first cervical and spinal accessory nerves. In one case the head was drawn back, and the spine bowed so that the patient was jerked into a squatting posture, the gastrocnemius being finally affected.

In other cases the spasms occurred when the individual began to walk. In still other cases there was a rhythmical motion when the patient attempted any simple voluntary action. These Weir Mitchell called "pendulum spasms," the number of twitches averaging 160 per minute, and recurring with great regularity.

Bamberger<sup>2</sup> reports a case which resembled spasm of another kind, Whenever the child was held in the standing posture his legs were drawn

<sup>1</sup> Am. Journ. Med. Sciences, Oct. 1876.

<sup>2</sup> Quoted by Handfield Jones, Functional Nervous Disorders.



up, and agitated by choreoid spasms, the spine and neck being twisted and contracted at the same time; but when he was placed upon his back these movements ceased.

### III. REFLEX SPASM.

Under this head may be classed a long list of local convulsive movements dependent upon a variety of causes. Sometimes there are worms in the intestinal canal, and at others a condition of irritability of the genitals; while peripheral irritations of many kinds enter into the etiology of the spasm.

I may illustrate the occurrence of one form of spasm by the following case:

I. A boy, 7 years old, seen at the request of Dr. Sayre, was well nourished, with rosy cheeks and well-rounded muscles of the upper extremities. His morbid condition had existed from birth, and he possessed a congenital phimosis, the prepuce being firmly fastened over the glans, and the preputial orifice was very small and surrounded by a rigid ring of toughened skin. On entering the room I was struck by the extraordinary restlessness and activity of the child. He was lying on the

Fig. 71.



Reflex Spasm from Genital Irritation.

bed, and his lower limbs were drawn up and agitated by irregular spasms. The arms were also convulsed, and their movements were distinctly choreic. When held upright the child was unable to stand, not from any paresis, but from the apparent loss of co-ordinating power, the legs becoming rigid, and the toes of both feet adducted, more particularly the left. The child was unable to speak, but attracted the attention of those around him by queer sounds. His face was distorted, just as we often see it in old choreic patients, but there was no evidence of imbecility. I did not infer that there was any mental trouble, except a preponderance of emotional disturbance, the boy being very fearful that he was to be hurt. Upon interrogating I found that he was quiet during sleep, that his appetite was good, and that there was no irregularity or disturbance of the functions of the bowels or bladder. The penis was not so sensitive as I had expected to find it from Dr. Sayre's description of previous cases. Titillation did not produce immediate erection, nor any increase of the spasmodic movements. On taking him upon my lap the thighs and legs were immediately drawn up; there was no evident pain produced by pressure on the spine.

A form of reflex spasm of the eyelids was reported by Von Gräefe,<sup>1</sup> which rendered the patient helpless, for he was unable to go about alone. There was no pain produced on pressure in the course of the fifth nerve; but when pressure was made on the glosso-palatine arch on the left lower jaw, the spasm ceased at once, and the patient could open his eyes. A putrid ulcer was found at this locality, which acted as a centre of irritation upon the gustatory nerve.

#### IV. FACIAL SPASM WITHOUT PAIN.

A form of facial spasm not connected with voluntary motorial movement is occasionally met with, the orbicularis palpebrarum or buccinator being affected alone, or all the muscles of the face supplied by the portio dura being convulsed. The trouble differs from epileptiform tic for the reason that it is unaccompanied by pain. I have been so fortunate as to see two of these cases. One was that of a gentleman aged 56, who suffered an almost constant spasm of the orbicularis of the eye, which was always increased when he was fatigued. The eye would become red, and there was usually a discharge of tears, which were unable to find their way into the lachrymal duct, and consequently ran on the cheek. Cases of unilateral painless spasm have been reported.

#### V. TORTICOLLIS.

The sterno-cleido mastoid muscle may be the seat of a spasmodic contraction. This condition may be preceded by peripheral trouble, such as painful dentition, which was the cause in one of Romberg's cases, or by such general disease as rheumatism. One case, which was seen by Dr. White and myself, was preceded by chorea, and another, that I saw at the New York State Hospital for Diseases of the Nervous System, was due to general anæmia. In both these cases, as well as in others I have observed the head was bent forward and the chin pulled downward. In one case, that of the elderly woman at the Hospital, the spasms were intermittent. Radcliffe reports a case which somewhat resembles this. The muscles of the neck were tender and the seat of soreness, and the movements were attended by pain. The spasms are usually increased by emotional excitement, but subside during sleep. The notes of my case are the following:—

M. A. A., aged 56, U. S. Came to the hospital Oct. 29, 1872. Her present trouble began five years ago in a very gradual manner. There are now marked clonic spasms of the muscles of the anterior part of the left side of the neck. With their intermitting contraction, there is some pain at the lower insertion of the sterno-cleido-mastoideus muscle; the trapezius is also the seat of spasmodic contraction. There is headache, and pain at the upper part of the cord. Patient's expression anxious and excited. Galvanism to muscles and spine, and zinci phosphidi gr.  $\frac{1}{2}$  t. i. d. Patient complains of dizziness and constipation.

<sup>1</sup> Schmidt's Jahresbericht, vol. 127, p. 30; reported by H. Jones, p. 390.

The muscles concerned in this form of disease are the sterno-cleido-mastoideus, complexus, trapezius, and levator anguli scapulæ.

**Pathology.**—Weir Mitchell has divided the conditions under which spasms of this kind may occur into three groups :—

1. "Those in which the functional activity of a muscle or set of muscles gives rise at times to an exaggeration of the motion involved naturally, and sometimes also to a more or less spasmodic activity in remoter groups.

2. "Those in which the functional action of one group results only in sudden and possibly in prolonged acts, tonic or clonic, in remote groups of muscles not implicated in the original movement.

3. "Those in which standing or walking occasions general and disorderly motions affecting the limbs, trunk, face, and giving rise to a general and uncontrollable spasm without loss of consciousness."

The central condition is one of great reflex irritability; certain forms of repeated irritation producing an activity of the motor centre which results in an abnormal increase in reflex susceptibility.

**Treatment.**—Agents which lower the excitability of voluntary muscular action are to be adopted. Among these hyoscyamia, gelseminum, musk, ether and assafoetida are efficient when used cautiously. Rest, and removal of the peripheral irritation, should the spasm be of reflex origin, and the ether spray to the spine, are to be resorted to; and at the same time various measures which improve the individual's general condition are in order. If all of these drugs I have mentioned be powerless to subdue the excitable condition of the muscles, I prefer profound brominization, which sometimes controls the movements. Myotomy in torticollis has not proved itself to be a successful operation, and so I do not recommend it. In other conditions, such as adherent prepuce, an operation is the only method that promises a cure.

The use of electricity in spasmodic affections is to be resorted to as promptly and thoroughly as possible. In torticollis it has hitherto been only moderately beneficial.

The lack of uniform success in the cases reported and a realization of the fact that electricity is of such great use in so many other spasmodic affections leads me to believe that many more patients might be relieved if the treatment were directed with a view to meet the pathological indications, which after all seem plain enough. In the early stages, it appears that the anterior muscles of the neck are not primarily affected, but rather the trapezius, and at such a stage the electrization of the sterno-mastoideus seems unwise. In other cases the approximative galvanization of the spinal accessory is indicated, while in the confirmed cases, which by the way we see the most of, I am about to speak of a treatment which I am not aware has been described heretofore. I find no allusion to the simultaneous employment of the two currents for the production of their physiological effects.

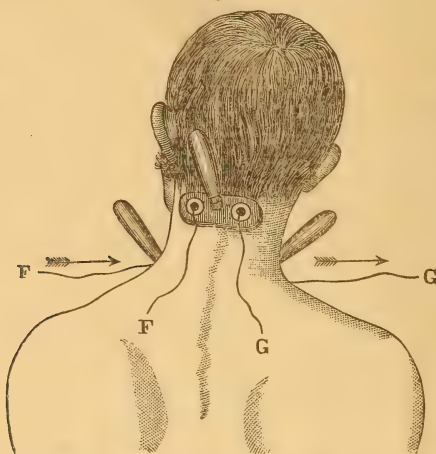
In the early part of 1879 my attention was first called to their use by a patient who had been under the care of my friend Dr. Findlay, of Ha-



vana, and who had been greatly relieved. Knowing nothing of Dr. Findlay's plan of treatment, I began a series of experiments to determine the best form of application and electrode, and after some trouble devised a method.

An electrode was constructed, which is armed with two sponge-covered pads, one of which is connected with the *positive* pole of a galvanic battery of twenty cells, while the other is attached to the *negative* wire of an induction coil. The double electrode is to be applied at the back of the neck, the two plates forming the terminal ends of the galvanic and faradaic apparatus, and being insulated by a central plate of hard rubber. Any ordinary double electrode may be used, however, and will answer every purpose. The *negative* galvanic electrode is to be placed over the insertion of the sterno-cleido-mastoid muscle of the *affected* side, so that a descending current is sent through the contracted muscle, while upon the insertion of the muscle of the other side is placed a sponge-covered electrode attached to the *positive* wire of the induction coil. The antagonistic muscle is thereby subjected to the stimulation of an ascending current from the faradaic apparatus.

Fig. 72.



The treatment of these cases is suggested entirely by the physiological influence of the two currents upon muscular tissue. In wry-neck of the spastic variety there is of course on one side a condition of tonic spasm, while on the other side the antagonistic muscle is necessarily in a condition of lowered tone, subjected as it is to the strain imposed by the position of the head and by the unavoidable traction. It will be seen that the condition of the antagonist is worse even than that of an opposing muscle in some other part of the body where there is less mechanical strain or tension of parts, as in this case the weight of the head is a factor in the disease which prevents the opposing muscle from ever being properly subjected to the improving influence of treatment.

A paralysis unaccompanied by contractures, and consequently

with no permanent stretching of opponents is, as we well know, much more readily improved by electricity if the strain be removed by proper appliances—such, for instance, as the apparatus devised by Van Bibber and Detmold for lead and facial paralysis. In the case of wry-neck, it must be borne in mind that, as no apparatus can be suggested which will do more than tire out the vicious spasm of the contracted sterno-cleido-mastoideus (a therapeutical measure which I consider to be unphysiological, from the fact that the spasm is an evidence of deficient or irregular innervation), a procedure which will tend to diminish the irritability of the muscle in spasm, while increasing the energy and improving the nutrition of the weakened opponent, is by far preferable.

In many cases, I am convinced, there is an hysterical element, which is decidedly increased by forcible restraint; and that this feature of the trouble belongs both to men and to women, I have no doubt. It is not difficult to imagine that harsh or irritating treatment will do harm in such cases.

In the varieties of wry-neck connected with disordered movements, there are several methods of treatment in vogue, which are sometimes successful. The ether-spray, either mediate or immediate (in the one case applied to the back of the neck; in the other, to the muscles themselves for five minutes at a time), does good in some cases. In other cases the local injection of sulphate of atropia will markedly modify the spasm, while, in cases of great severity, decided doses of the tincture of gelsemium sempervirens or of hyoseyamia will diminish the violence of the spasmodic condition. A case mentioned by Radcliffe was treated with hypodermic injections of Fowler's solution, and improved somewhat.

While I am not disposed to take the grave view of the prognosis expressed by Reynolds, it must be confessed that there are very many examples which are not permanently benefited. Under this head come those which are unquestionably varieties of spinal or cerebral sclerosis. I have seen a case of progressive muscular atrophy which had been mistaken for wry-neck. In cases of organic disease of the brain, the early history of the case and the connection perhaps with paralysis or contracture of the extremities show us that the case is not one of true torticollis. Exceedingly rare cases of tonic contraction are met with in which the essential condition is dislocation or disease of the cervical vertebræ. Then, of course, the prognosis is bad.

The cases most readily helped are those dependent upon rheumatism or hysteria, and in such the prognosis is highly favorable. In the latter form of trouble, one or two applications of the faradaic current are alone sufficient, and, if the diagnosis is certain, it will be found that a shower of sparks, derived from a Holtz machine, directed upon the muscle, will favor a sudden disappearance of the spasm.

A case of clonic spasm of the facial muscles of a very serious and persistent nature was cured by Baum, by nerve section. A slight paralysis of half an hour's duration was produced.<sup>1</sup>

<sup>1</sup> Berliner Klin. Woch., 1878, No. 40, and Bost. Med. and Surg. Journal, Sept. 4, 1879, p. 341.

## PROFESSIONAL CRAMP.

**Synonyms.**—Writer's cramp, Dancer's cramp, Telegrapher's cramp; Dyskinésie professionnelle; Melker-krampf, Schuster-krampf, Nähekrampf.

This very interesting condition, which follows the overtraining of groups of muscles, is found among all who engage in occupations which require the exercise of particular voluntary muscles of the upper and lower extremities to an excessive degree. Among these individuals such protracted muscular action, especially when of a delicate kind, is likely to be followed by spasmodic movements such as would come under the first group of Mitchell.

It is the first of the above varieties that at present interests us the most.

WRITER'S CRAMP is the form of hyperkinesis with which we are the most familiar, and it is difficult to fail in recognizing its true character. After continued and fatiguing use of the pen the hand may become at first tired; afterwards the patient suffers from sharp pains which run from the hand up the arm, while dull pains seated in the ball of the thumb, the dorsal aspect of the fingers, the wrist, or at the exposed portion of the ulnar nerve at the elbow, are to be found as well. His first intimation may be a certain tired feeling, or, as a very intelligent patient under my care expressed it, "The first idea of my trouble came from the feeling that I *had an arm*. My mind was directed to it, and whether resting or at work, it felt like a clumsy part of my body." If the individual carefully forms his words, or if he "writes with his fingers"—a habit which schoolboys have, and which sometimes continues through life—the trouble is much more probable than when he uses his whole hand in guiding his pen. He may find after a while that when he attempts to write, the hand will fly upwards as the result of a spasm of the extensors and other muscles on the dorsal and ulnar side of the forearm, so that it is often impossible to form more than one or two words of a note before the trouble begins.

This impaired writing power may exist to a lighter degree; but when the individual persists in his attempts, the convulsion is certain to take place. A light tonic spasm of the abductor *mimimi* *digiti* may occur when the little finger is separated from its fellows, and this is sometimes an early sign of the disease. He may educate the left hand to do the work of the right, and after a while may learn to use it in a satisfactory manner; but very soon this too becomes affected, and he can write with neither hand. Other muscular movements are freely performed, and even some which closely resemble that of holding the pen. Trembling sometimes supervenes, while fibrillary muscular contractions are suggestive of the confirmed disease. As is the case in sclerosis, the disorderly movements, or the spasms, seem to be intensified when the patient attempts to write in the presence of a looker-on, and he usually makes sad work.



The fingers, forearm, and wrist sometimes become the seat of lost power, and this is marked in the three first fingers of the right hand, and the pronators and supinators lose power. Sensation is rarely lost or impaired. In some cases the flexors of the hand and the small muscles of the thumb are so weak that the point of the pen cannot be kept in contact with the paper, as the extensors seem to act independently.

The same form of cramp affects the thumbs and fingers of telegraphers, so that their work eventually becomes an impossibility. Onimus<sup>1</sup> presents a case. A telegraphic operator, 19 years of age, first experienced difficulty in making dots; "d" was made better than "u;" and it was found that when a line was first the dots were more easily made; but letters like "h" or "p" were exceedingly difficult.<sup>2</sup>

Dancers' cramp has also been observed. Schultz<sup>3</sup> describes this form of disease, of which he has seen three cases. It affects the solo dancers of the ballet as a rule, and the history of one case was the following:—

"The patient complained of suffering very severe pains while dancing. Beginning in the soles of both feet, the pains spread with increasing severity to the calves of the legs; they at last became so violent that her feeling of security was lost, the feet seeming as if made of wood. These pains were accompanied with violent palpitation; and, if she continued to dance, she felt faint and sometimes lost consciousness, the body becoming quite rigid. When the pain and palpitation were less intense, the pain continued after dancing, and ceased very gradually, leaving some tenderness of the soles; on attempting again to dance the suffering would recur again. Dr. Schultz found, from the examination of these cases, that the cause of pain lay in the *pas* performed on the points of the feet, and is owing to exhaustion of the muscles which fix the metatarsus and phalanges of the great toe. The shoe worn by the dancer, without which the ballet step seems to be impossible, is made as follows: The dancing-shoe is made rather wide; the sole is of soft leather, and shorter than the foot, reaching only as far as the posterior third of the ungual phalanx of the great toe. The upper part, generally of satin, projects forward, and supplies the place of the deficient leather of the sole. This part of the satin is worked threads, so that it may not be torn. In the interior of the shoe, over the leather sole, is a layer of thin, firmly-pressed pasteboard, either extending over the whole breadth of the anterior part, or limited to the length of the great toe. In the former case it is carried back, gradually narrowed as far as the heel. The leather sole and its covering are lined with fine kid leather. The heel part of the shoe is quite soft, consisting only of satin; and the shoe is fastened above the ankle by narrow ribbons. Without this preparation the pointed step is impossible."

I have met with the affection among violin-players, and within the past year have had a patient under treatment. He had been diligently practising a "run," which involved the necessity of complicated movements of

<sup>1</sup> Gaz. Méd. de Paris; Chicago Journal of Mental and Nervous Diseases, July, 1875.

<sup>2</sup> (- - - u) (— - - d) (--- h; ----- p.)

<sup>3</sup> Wiener Med. Woch.

the fingers; and it was his custom, on arising in the morning, to spend a half hour or so in playing the difficult passage; and on the day of the concert he worked for several hours at the same task, but upon attempting to play in the evening he found it utterly impossible to do so, as his fingers would become rigid and refuse to obey the will. It was some months before he could again play.

Onimus,<sup>1</sup> in describing a form of impaired power and consequent muscular atrophy, which he calls "professional muscular atrophy," details a case which resembles somewhat the form of functional disease which we are considering. It begins by muscular cramp, and there is subsequent loss of power with wasting. I therefore think we may consider this affection as a connecting link between scrivener's cramp and progressive muscular atrophy. He says:—

"Recently I observed one case which it was most difficult to differentiate from progressive muscular atrophy, as the atrophied muscles were the same as those which are the first affected by this latter affection. They were the muscles of the thenar eminence, and chiefly the adductor pollicis. The patient was an enameller, who had to hold an object all day between his thumb and index finger. He first got cramps in the thumb, which suggested the idea of scrivener's palsy; then tremor of the thumb, on account of the fibrillary contractions; and, lastly, atrophy. Under the influence of treatment there was a rapid amendment, which showed that the case was really one of professional muscular atrophy, and not commencing progressive atrophy."

**Causes and Pathology.**—This spasmodic affection follows the continued use of the muscles which are concerned in delicate muscular actions; and is not only produced by writing, but, as I have shown, by other forms of manipulation requiring great delicacy of co-ordination. The higher and the more complex is the character of these acts, and the more easily the faculty to perform them becomes developed, so much the greater is the danger of the disease. An act which requires at first mental direction of a superior kind, when acquired and executed unconsciously, is much more likely to give rise to this neurosis than one of a grosser kind, or one which is constantly performed under the active direction of the will. For this reason writer's cramp is much more rare among those who write and meanwhile compose, than among clerks or copyists who do "machine work." Constant use of the pen of this kind is seen to be followed by mischief. Such causes as piano-playing or violin-playing are by no means rare. A young lady, sent to me by my friend Dr. D. M. Stimson, owed all her trouble to a bad habit she had contracted of reading novels while she practised her scales. In her case there was extensor paralysis, and some loss of sensation, which remained after a spasmodic stage.

The conditions then, with the exception of paralysis, are the result of an over-developed automatism, and are not, I am convinced, connected

<sup>1</sup> London Lancet, Jan. 22, 1876.

with any central change, though Mr. Solly<sup>1</sup> is inclined to consider that there is degeneration of the motor cells in the upper part of the cord.

In writing a familiar word, or collection of words, the educated individual does not stop to form every letter, but the pen is unconsciously guided. It is even possible to talk while writing or playing the piano, and equally complex feats are performed while the mind is not engaged. In many of these acts the volition is directed in other channels, or is behind the muscular action. The pen travels in advance of the mind; and should this state of things be so exaggerated as to become more than a phase of the ordinary automatism which enters into the performance of many of the functions of daily life, there remains condition of disordered and heightened activity which is uncontrolled by the will, and is symptomatized by the spasms of which I have spoken. A more advanced condition consists in exhaustion of the motor cells at the upper part of the cord, and as a result we find loss of power and occasionally atrophy. Poore<sup>2</sup> does not believe in the central organic origin of the disease; but Solly,<sup>3</sup> Smith,<sup>4</sup> and others take this view of the case.

Among 24 cases which I have seen, the occupation of the individuals was as follows:—

|                   |    |                      |   |
|-------------------|----|----------------------|---|
| Clerks . . . .    | 14 | Stenographer . . . . | 1 |
| Engraver . . . .  | 1  | Musicians . . . .    | 3 |
| Lawyers . . . .   | 2  | Type-setter . . . .  | 1 |
| Clergymen . . . . | 1  | Cigar-maker . . . .  | 1 |

The patients were all men but one, and with this exception were between the ages of 30 and 60; I do not believe, however, this latter fact has very much importance.

**Diagnosis.**—Progressive muscular atrophy may be mistaken for the paralytic form, but when it is remembered that the paralysis precedes the atrophy (should such tissue-change take place), and that progressive muscular atrophy is rarely so limited, there is no reason why the real nature of the trouble should not be recognized. Neuralgia of the cervico-brachial variety is a common symptom, and its real significance may not be detected; the subsequent element of spasm, tremor, or paralysis will, however, remove any doubt from the mind of the observer.

**Prognosis.**—If the individual gives up the occupation which has produced the affection, there is no reason why he should not recover, provided the disease has not become confirmed, and even in this form Jaccoud<sup>5</sup> speaks of a rare temporary amelioration. It has been my experience that, if taken in hand promptly, the patient may be cured. Sixteen of these cases were absolutely cured, and continued so as long as

<sup>1</sup> Surgical Experiences, London, 1865, p. 205.

<sup>2</sup> Practitioner, June, July, and August, 1873.

<sup>3</sup> Op. cit.

<sup>4</sup> Lancet, March 27, 1869.

<sup>5</sup> Op. cit., p. 302.



they refrained from their work. Two were improved, but upon beginning the pursuit of their calling had relapses. The remainder were of the paralytic variety, and have been for some time under treatment.

**Treatment.**—Rest and electricity are the means at our command. A galvanic current is found to be the most beneficial, and the electrodes should be so small as to include but one muscle at a time in the circuit. The current must be mild, or it will only aggravate the disease. Besides this application to special muscles, one pole may be placed at the nape of the neck, and the other to the muscles of the hand and forearm.

A. W., aged 38. The patient had followed the occupation of clerk for several years, and had assiduously worked at his desk for many hours in the day. Two weeks before I saw him he noticed an impairment in his writing power, and this consisted in an inability to write without the occurrence of a convulsive contraction of the extensors of his right forearm, by which the pen flew from the paper. This did not occur at the moment of writing, but after a few words had been finished. He tried to keep the hand steady by the influence of the will, but all his efforts were ineffectual. When he attempted to hold the point of any small object, such as a stick or pencil, against the surface, the same spasm would occur. There was no wasting of the muscles, pain, or other symptom. I determined to try galvanism combined with manual exercise, and the internal application of strychnia in doses of  $\frac{1}{4}$ th of a grain. Galvanization of the flexors of the forearm and of the small muscles of the hand was made, and, at the same time, the positive pole was held for a few minutes at the nape of the neck. He was directed to procure the rounds of a chair with which to exercise. Galvanization was persevered in, although the progress was very slow. At first he could not write more than two words (almost illegibly); but as he grew better, these spasms disappeared.

Three *séances* a week kept up for a period of about three months effected such an improved condition that he was finally discharged at the end of that time.

Strychnia and iron, or conium, are remedies which may be used in conjunction. The ether spray apparatus does great good, and I have occasionally benefited my patients by fastening the hand in an immovable apparatus or splint. Absolute cessation of the particular work which gave rise to the malady is *to be insisted upon*, and no benefit will result from any form of treatment unless this command of the physician is respected.

When the patient attempts writing anew he should provide himself with a pen having a cork holder, and this may be purchased from any good stationer. He should change his system of penmanship and acquire the so-called free hand style, in which the fingers are engaged only in holding the pen, and the other motions are performed by the muscles of the forearm. The attempt at "shading" the lines should not be made but he should endeavor to adopt the round hand and avoid "pot hooks" and "up and down" strokes as much as possible.

Sea air, salt baths, and a change of habits and scene are all fraught with benefit.

I do not consider tenotomy advisable except in extreme instances.

### ŒSOPHAGISMUS.

A comparatively rare neurosis often met with among women consists in a spasmodic contraction of the œsophagus. It is usually hysteroid in character, or may be the reflex result of a simple stomatitis, beginning, perhaps, in a trivial irritation of the food passage; and giving the individual little annoyance at first, it may develop into a condition causing great misery and suffering from dysphagia, so that she may be unable to swallow anything but fluids, and these in small quantities, and most easily when they are warm.

"Tightness of the throat," the globus hystericus and, more or less, hyperæsthesia, may be symptoms which precede or accompany the trouble.

There is emotional derangement as well, and the patient weeps and is despondent. The symptoms of spinal irritation may or not be manifested, and there is usually some spinal tenderness. A patient sent to me by Dr. Cohen, of Philadelphia, had suffered for several years, and I have examined other patients who have suffered even longer. The discomfort attending the local trouble affects the general condition, and malnutrition from insufficient food and sleeplessness reduce the patient in every way. An examination, by means of an olive-pointed bougie, will immediately apprise us of the cause of the annoyance, and among hysterical women, who complain of their inability to swallow, we will often find, by local examination, that there is a true œsophageal spasm, which is sufficient to account for the subjective expressions some of us are inclined to disregard.

I have met with subjects who complained of a spasm of the upper part of the pharynx with sharp pain, and in several instances have traced its origin to the immoderate use of tobacco.

**Treatment.**—The affection is a troublesome and persistent one. Galvanization of the sympathetic; local treatment by bougies and ether spray to the back of the neck are important external remedies; while we may give internally, hysocyamia or any of the anti-spasmodics before alluded to.

THE END.





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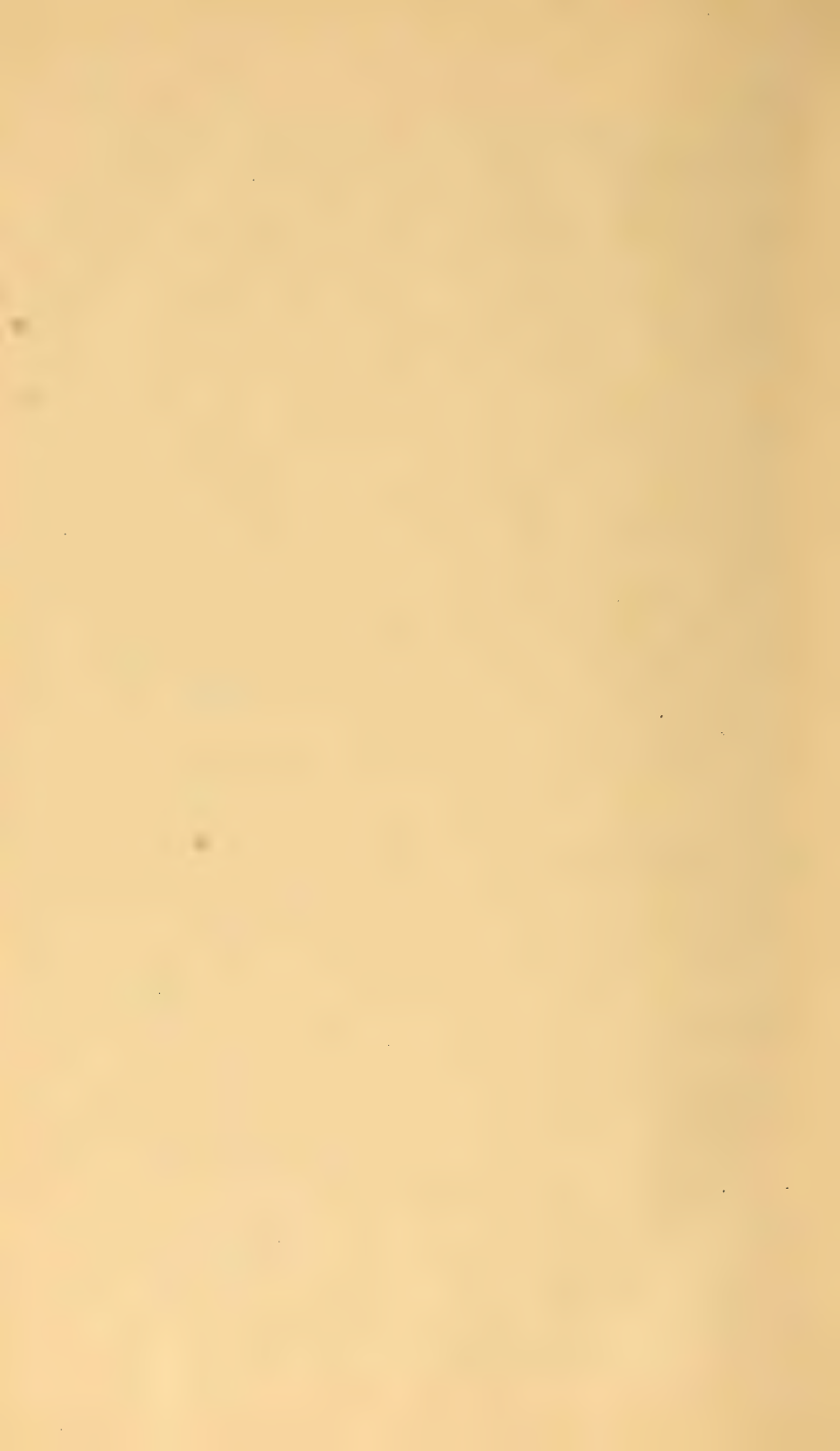
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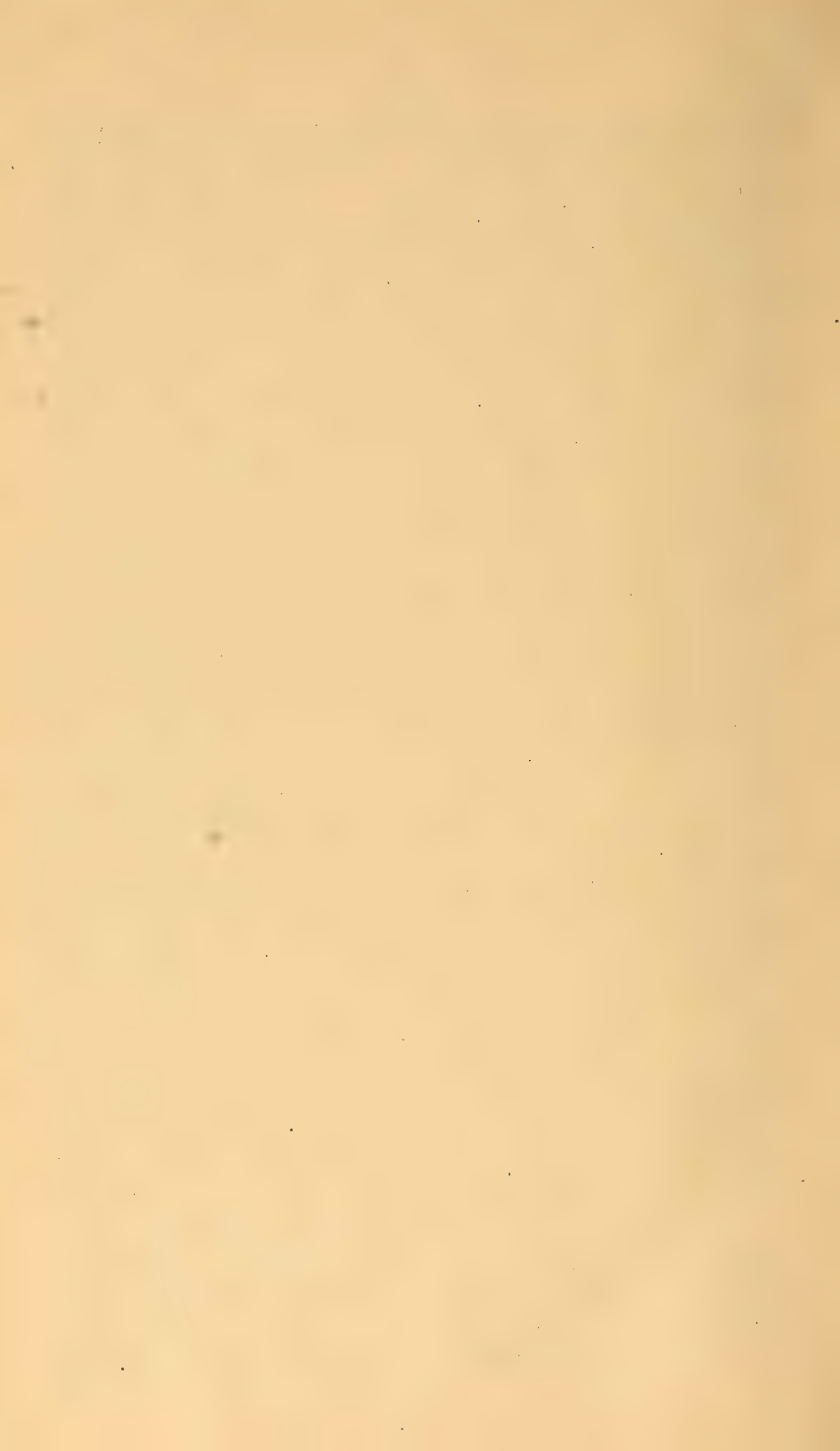
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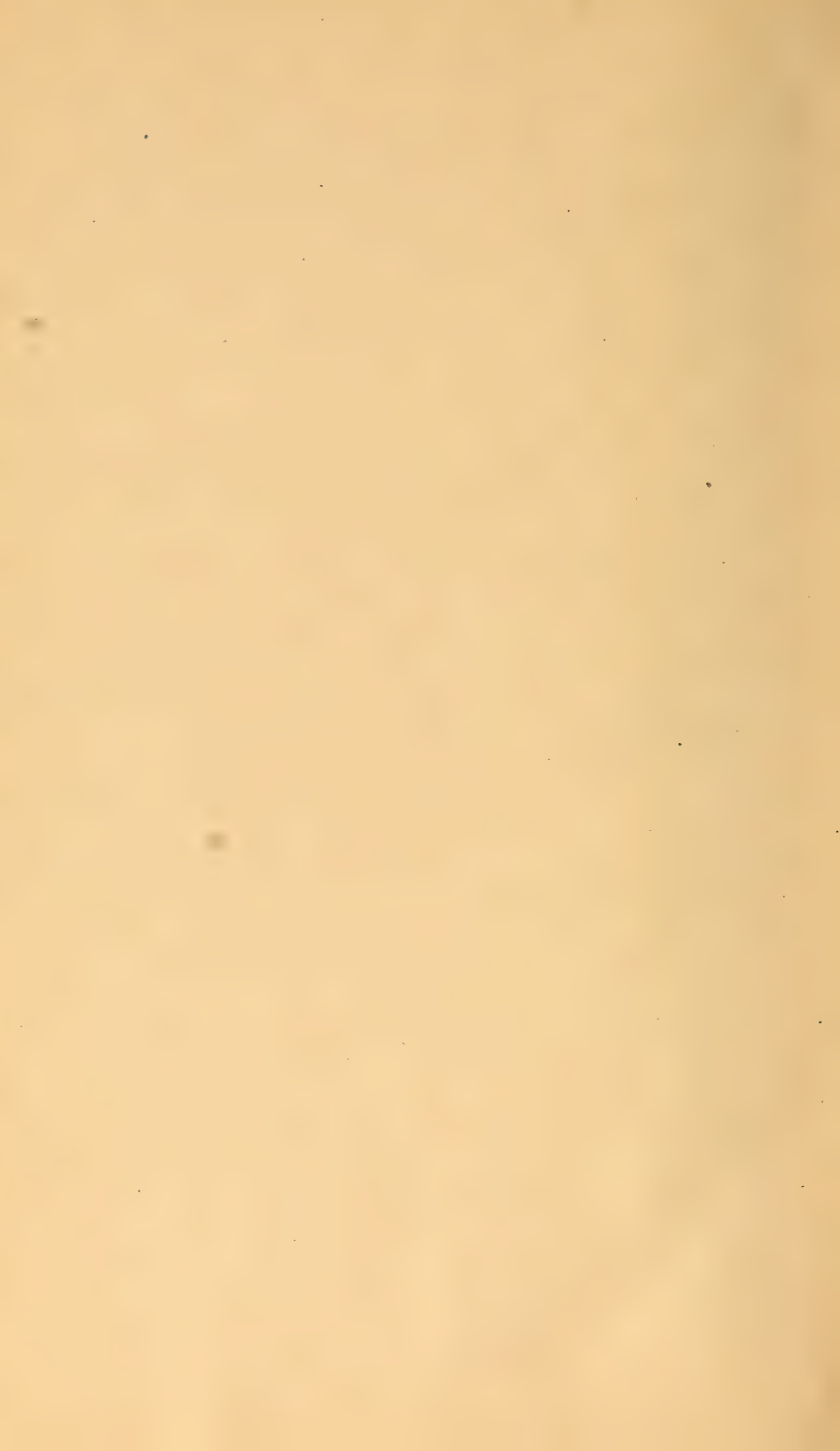






















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